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THE TRANSACTIONS

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VOL. XXV.—NEW SERIES

SESSION 1905-1906

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PREFACE

THE present Volume is the *Twenty-fifth* of the *New Series*, and contains a record of the work done during the past Session.

That work, as hitherto, embraces the communication of Original Papers; the exhibition of Patients, illustrating rare and interesting forms of disease; and the exhibition of Pathological and other Specimens, so essential to the proper understanding of the morbid changes which take place in the human body.

During the past Session Extra Meetings were held for the exhibition of Patients, Pathological Specimens, Instruments, etc. It is hoped that such Meetings will materially increase the usefulness of the Society.

One of the Clinical Meetings was held in the Royal Infirmary, which was very successful.

It is believed that the publication of the Transactions in this permanent form will prove a valuable contribution to medical literature, will encourage the Members to take a more active part in the work of the Society, and will tend in no small degree to increase the influence and usefulness of the Medico-Chirurgical Society of Edinburgh.

A general Index to the first Twenty Volumes of the New Series was published in 1902. It is uniform with the other Volumes.

WILLIAM CRAIG,
Editor.

September 1906.

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10	Dr F. L. Atkinson, 15 Merchiston Place,	1904
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	Dr J. W. Ballantyne, 24 Melville Street,	1885
	Dr A. H. Freeland Barbour, 4 Charlotte Square,	1881
	Dr James M. Beattie, 8 Thirlestane Road,	1904
15	Dr Lewis Beesley, 21 West Hill, St Leonards-on-Sea,	1905
	Dr J. H. Montgomerie Bell, East Morningside House, Clinton Road,	1905
	Joseph Bell, Esq., 2 Melville Crescent,	1862
	Dr G. A. Berry, 31 Drumsheugh Gardens,	1883
	Dr W. T. Black, 2 George Square,	1877
20	Dr Robert H. Blaikie, 10 Mayfield Gardens	1883
	Dr Bleloch, 26 Gilmore Place,	1871
	Dr G. P. Boddie, 73 Bruntsfield Place,	1888
	William Booth, Esq., 2 Minto Street,	1888
	Dr John M. Bowie, 6 Hope Park Crescent,	1901

		Date of Admission.
25	Dr F. D. Boyd, C.M.G., 22 Manor Place,	1891
	Dr Byrom Bramwell, 23 Drumsheugh Gardens, *Vice-President*	1876
	Dr Edwin Bramwell, 23 Drumsheugh Gardens,	1900
	Dr N. T. Brewis, 23 Rutland Street,	1886
	Dr Henry Grey Brown, 1 Cluny Avenue,	1905
30	Dr J. J. Graham Brown, 3 Chester Street,	1878
	Dr J. Murdoch Brown, 9 Walker Street,	1885
	Dr Alexander Bruce, 8 Ainslie Place,	1883
	Dr A. H. Buchan, 2 Dean Terrace,	1899
	Dr Buist, 1 Clifton Terrace,	1877
35	Dr T. M. Burn-Murdoch, 14 Charlotte Square,	1886
	Dr Cadell, 22 Ainslie Place,	1870
	Francis M. Caird, Esq., 13 Charlotte Square,	1883
	Dr H. L. Calder, 60 Leith Walk, Leith,	1884
	Dr James Cameron, 13 Fettes Row,	1895
40	Dr Malcolm Campbell, 17 Walker Street,	1904
	Dr A. N. S. Carmichael, 6 Morningside Road,	1902
	Dr Edward Carmichael, 21 Abercromby Place,	1887
	Dr E. W. Scott Carmichael, 32 Rutland Square,	1901
	Dr George Scott Carmichael, 25 Braid Road,	1903
45	Dr J. Carmichael, 22 Northumberland Street,	1870
	Dr George J. R. Carruthers, 4A Melville Street,	1901
	C. W. Cathcart, Esq., 44 Melville Street,	1883
	Dr J. G. Cattanach, 3 Alvanley Terrace,	1895
	Dr T. F. S. Caverhill, 6 Manor Place,	1883
50	Dr George L. Chiene, 23 Alva Street,	1901
	Professor John Chiene, C.B., 26 Charlotte Square,	1867
	Dr Church, 36 George Square,	1876
	Dr Clouston, Tipperlinn House, Morningside Place,	1861
	Dr John D. Comrie, 7 S.-E. Circus Place,	1902
55	J. M. Cotterill, Esq., 24 Manor Place,	1878
	Dr John Craig, 71 Bruntsfield Place,	1900
	Dr William Craig, 71 Bruntsfield Place,	1869
	Dr D. M. Crichton, Burgh House, Maryhill, Glasgow,	1906
	Professor Sir John Halliday Croom, 25 Charlotte Square, *Vice-President*,	1870
60	Dr G. Matheson Cullen, 50 Minto Street,	1892
	Dr John Cumming, 20 Gilmore Place,	1894
	Professor D. J. Cunningham, 18 Grosvenor Crescent,	1903
	Dr T. B. Darling, 13 Merchiston Place,	1887
	Dr G. D. Darlington, 10 Ardmillan Terrace,	1898
65	Dr M. Dewar, 24 Lauriston Place,	1885
	Dr J. T. Dickie, 37 Lauriston Place,	1900
	Dr George Dickson, 9 India Street,	1884
	Dr George Dickson, 14 Ardmillan Terrace,	1901
	Dr W. E. Carnegie Dickson, 9 India Street,	1902
70	Dr Frederick Dittmar, 26 Heriot Row,	1906
	Dr C. E. Douglas, Cupar-Fife,	1894
	Dr Kenneth M. Douglas, 19 Chester Street,	1888
	Dr W. B. Dow, Dunfermline,	1879
	Dr J. W. Dowden, 27 Walker Street,	1893
75	Dr W. B. Drummond, 9 Brougham Place,	1900
	Dr James K. Drysdale, 22 Coltbridge Terrace,	1901
	Dr Henry J. Dunbar, 1 Kew Terrace,	1906
	Dr John A. H. Duncan, 32 Morningside Drive,	1895
	Dr Kirk Duncanson, 22 Drumsheugh Gardens,	1871
80	Dr H. M. Dunlop, 20 Abercromby Place,	1883
	Dr J. C. Dunlop, 33 Chester Street,	1892
	Dr J. Dunsmure, 53 Queen Street,	1872
	Dr John Eason, 196 Newhaven Road,	1900
	Dr C. C. Easterbrook, Glengall, Ayr,	1899
85	Dr William Elder, 4 John's Place, Leith,	1892

		Date of Admission.
	Dr J. Haig Ferguson, 7 Coates Crescent,	1885
	Dr Thomas Finlay, 7 Mary's Place,	1903
	Dr W. A. Finlay, 50 Trinity Road,	1875
	Dr Andrew F. Fleming, St John's Road, Corstorphine,	1905
90	Dr R. A. Fleming, 10 Chester Street,	1890
	Dr A. B. Flett, 60 George Square,	1903
	Dr A. Dingwall Fordyce, 19 Coates Crescent,	1901
	Dr J. S. Fowler, 68 Northumberland Street,	1895
	Dr John Fraser, 13 Heriot Row,	1878
95	Dr J. Hossack Fraser, Fernfield, Bridge of Allan,	1895
	Dr J. S. Fraser, 13 Heriot Row,	1905
	Prof. Sir Thomas R. Fraser, 13 Drumsheugh Gardens,	1865
	Dr W. Ernest Frost, 6 Atholl Place.	1904
	Dr F. Gardiner, 9 George Square,	1900
100	Dr Garland, 53 Charlotte Street, Leith,	1873
	Dr A. Campbell Geddes, 2 Rutland Square,	1904
	Dr J. H. Gibbs, 7 Coates Place,	1905
	Dr G. A. Gibson, 3 Drumsheugh Gardens,	1880
	Dr A. B. Giles, 4 Palmerston Place,	1893
105	Dr Alexander Goodall, 7 Mayfield Road,	1901
	Dr D. J. Graham, 26 Rutland Street,	1897
	Dr F. M. Graham, 16 Mayfield Gardens,	1894
	Dr J. Taylor Grant, 3 Manor Place,	1898
	Dr J. Allan Gray, 107 Ferry Road,	1879
110	Dr John L. Green, 23 Minto Street,	1904
	Professor Greenfield, 7 Heriot Row, *Vice-President*,	1886
	Dr G. L. Gulland, 11 Chester Street, *Secretary*,	1888
	Dr James A. Gunn, Materia Medica Department, University of Edinburgh,	1906
	Dr A. C. Guthrie, 21 Pilrig Street,	1890
115	Dr William Guy, 11 Wemyss Place,	1890
	Dr William Haldane, Viewforth, Bridge of Allan,	1889
	Dr George Halley, 7 Airlie Place, Dundee,	1902
	Dr D. Berry Hart, 13 Charlotte Square,	1886
	Dr James Harvey, 7 Blenheim Place,	1893
120	Dr Henry Hay, 11 Great King Street,	1884
	J. W. B. Hodsdon, Esq., 6 Chester Street,	1883
	Dr George Hunter, 33 Palmerston Place,	1876
	Dr J. Hutcheson, 44 Moray Place,	1890
	Dr R. W. Inkster, 16 Windsor Street,	1895
125	Dr Alex. James, 10 Melville Crescent,	1877
	Dr Allan Jamieson, 35 Charlotte Square,	1876
	Dr Hugh Jamieson, 13 Lauriston Place,	1889
	Dr J. Boyd Jamieson, 43 George Square,	1900
	Dr G. M. Johnston, 7 Wellington Place, Leith,	1888
130	Dr R. J. Johnston, 1 Buccleuch Place,	1897
	Dr R. M'Kenzie Johnston, 2 Drumsheugh Gardens,	1883
	Dr C. Kennedy, 5 Salisbury Road,	1886
	Dr C. B. Ker, City Hospital, Comiston Road,	1894
	Dr George Kerr, 6 St Colme Street,	1885
135	Dr J. H. A. Laing, 11 Melville Street,	1889
	Dr H. G. Langwell, 4 Hermitage Place, Leith,	1899
	Dr David Lawson, Nordrach-on-Dee, Banchory,	1901
	Dr H. A. Leebody, Corstorphine,	1899
	Dr Edwin M. Lithgow, 8 Alva Street,	1901
140	Dr John D. Lithgow, 8 Alva Street,	1901
	Professor Harvey Littlejohn, 1 Atholl Crescent, *Treasurer*,	1889
	Emeritus Professor Sir Henry D. Littlejohn, 24 Royal Circus,	1853
	Dr T. Davy Luke, 95 Shandwick Place,	1900
	Dr R. A. Lundie, 55A Grange Road,	1883
145	Dr W. Leslie Lyall, 8 Murrayfield Gardens,	1902
	Dr D. C. A. M'Allum, 11 Greenhill Gardens,	1900

		Date of Admission.
	Dr P. M'Bride, 16 Chester Street,	1879
	Dr Angus Macdonald, 9 Rutland Square,	1902
	Dr Stuart M'Donald, 197 Newhaven Road,	1901
150	Dr John M'Gibbon, 22 Heriot Row,	1899
	Dr C. W. MacGillivray, 15 Charlotte Square,	1877
	Dr A. M. MacIntosh, 30 East Claremont Street,	1904
	Dr G. Mackay, 74 Bruntsfield Place,	1878
	Dr George Mackay, 20 Drumsheugh Gardens,	1887
155	Dr T. C. Mackenzie, Morningside Royal Asylum,	1904
	Dr W. Leslie Mackenzie, 1 Stirling Road, Trinity	1895
	Dr P. Hume Maclaren, 1 Drumsheugh Gardens,	1868
	Dr Malcolm M'Larty, 7 Bellevue Place,	1900
	Dr John Macmillan, 48 George Square,	1897
160	Dr J. C. G. Macnab, The Towers, Dysart,	1904
	Dr Alexander H. Macpherson, Cliftonville, Blackhall,	1901
	Dr John Macpherson, 8 Darnaway Street,	1891
	Dr G. Douglas M'Rae, Morningside Royal Asylum,	1904
	Dr John MacRae, Lynwood, Murrayfield,	1893
165	Dr Alexander M. Malcolmson, St John's Road, Corstorphine,	1905
	Major D. G. Marshall, I.M.S., 25 Chester Street,	1905
	Dr A. A. Matheson, 41 George Square,	1901
	Dr Roderick M. Matheson, 33 Buccleuch Place,	1902
	Dr Edwin Matthew, 6 Hampton Terrace,	1900
170	Dr D. Menzies, 20 Rutland Square,	1878
	Dr A. Miles, 20 Walker Street,	1892
	Dr J. Mill, 26 N. Fort Street, Leith,	1886
	A. G. Miller, Esq., 8 Garscube Terrace,	1867
	Dr W. H. Miller, 51 Northumberland Street,	1895
175	Dr D. Milligan, 11 Palmerston Place,	1885
	Dr W. Morrison Milne, 10 Newington Road,	1905
	Sir Arthur Mitchell, 34 Drummond Place,	1859
	Dr John Mowat, 5 Hope Park Terrace,	1885
	Dr Claud Muirhead, 30 Charlotte Square,	1866
180	Dr W. H. Myers, Indiana, U.S.A.,	1906
	Dr H. Oliphant Nicholson, 20 Manor Place,	1900
	Dr John Orr, 6 Strathearn Road,	1895
	Dr W. Basil Orr, 13 Braid Road,	1893
	Dr G. Keppie Paterson, 19 Albany Street,	1889
185	Dr J. V. Paterson, 5 Castle Terrace,	1900
	Dr Simla Paterson, 147 Bruntsfield Place,	1904
	Professor D. Noël Paton, University of Glasgow,	1885
	Dr C. Balfour Paul, 17 Walker Street,	1904
	Dr C. M. Pearson, 17 Alva Street,	1902
190	Dr H. A. Peddie, 24 Palmerston Place,	1893
	Dr R. W. Philip, 45 Charlotte Square,	1883
	Dr J. H. Harvie Pirie, 13 Alva Street,	1905
	Dr John Pirie, 15 Ardmillan Terrace,	1902
	Dr Playfair, 5 Melville Crescent,	1874
195	Dr Frederick Porter, 65 Morningside Road,	1904
	Dr T. Proudfoot, 30 Lauriston Place,	1889
	Dr Harry Rainy, 16 Great Stuart Street,	1896
	Dr Douglas G. Reid, 16 Greenbank Terrace,	1905
	Dr James Ritchie, 22 Charlotte Square,	1873
200	Dr L. C. Peel Ritchie, 13 Castle Terrace,	1904
	Dr W. T. Ritchie, 9 Atholl Place,	1900
	Dr Robert Robertson, 26 Royal Circus,	1901
	Dr William Robertson, 11 Denham Green Terrace, Trinity,	1902
	Dr W. Ford Robertson, 9 Priestfield Road,	1893
205	Dr W. G. Aitchison Robertson, 26 Minto Street,	1892
	Dr R. M. Ronaldson, 17 Morningside Road,	1895
	Dr T. R. Ronaldson, 8 Charlotte Square,	187

		Date of Admission.
	Dr A. Aitken Ross, 8 Newington Road,	1902
	Dr William Russell, 3 Walker Street,	1884
210	Dr J. M. Rutherford, Morningside Royal Asylum,	1900
	Dr E. G. Salt, 59 George Square,	1895
	Dr James Scott, 43 Minto Street,	1895
	Dr Theodore Shennan, 95 Mayfield Road,	1897
	Dr W. C. Sillar, 46 George Square,	1900
215	Emeritus Professor Sir Alexander R. Simpson, 52 Queen Street,	1859
	Dr F. D. Simpson, 7 Kew Terrace,	1905
	Dr G. F. Barbour Simpson, 50 Melville Street,	1900
	Dr John W. Simpson, 15 Glanville Terrace,	1901
	Dr Arthur H. H. Sinclair, 5 Walker Street,	1900
220	Dr A. A. Scot Skirving, C.M.G., 8 Randolph Crescent,	1897
	Dr A. T. Sloan, 22 Abercromby Place,	1885
	Dr Herbert Sloman, 9 Murrayfield Avenue,	1906
	Dr Andrew Smart, 50 Moray Place,	1865
	Dr James Smith, 4 Brunton Place,	1891
225	Dr John Smith, 11 Wemyss Place,	1856
	Dr J. Cormack Smith, 9 Brunton Place,	1896
	Dr J. W. Somerville, 12 Abbotsford Road, Galashiels,	1905
	Dr Arthur J. de Spiganovicz, 1 Seton Place,	1905
	Dr J. W. Steven, 13 Manor Place,	1900
230	Dr John Stevens, 2 Shandon Street,	1892
	Dr Norman L. Stevenson, 7 Alva Street,	1902
	Dr J. S. Stewart, 15 Merchiston Place,	1885
	Dr Robert Stewart, 25 George Square,	1892
	Dr William Stewart, 146 Ferry Road, Leith,	1889
235	H. J. Stiles, Esq., 9 Great Stuart Street,	1889
	Dr John Strachan, Dollar,	1867
	Dr John W. Struthers, 5 Lynedoch Place,	1901
	Dr W. J. Stuart, 9 Chester Street,	1902
	Dr William G. Sym, 12 Alva Street,	1889
240	Dr W. Taylor, 12 Melville Street,	1871
	Dr W. Macrae Taylor, 12 Melville Street,	1903
	Dr C. H. Thatcher, 8 Melville Crescent,	1876
	Dr R. Thin, 25 Abercromby Place,	1890
	Mr Alexis Thomson, 39 Drumsheugh Gardens,	1887
245	Dr H. Torrance Thomson, 10 Blenheim Place,	1900
	Dr John Thomson, 14 Coates Crescent,	1887
	Dr George Thyne, 13 Brunswick Street,	1904
	Dr T. J. Thyne, 16 Randolph Crescent,	1898
	Dr John Tod, 69 Ferry Road, Leith,	1895
250	Sir John Batty Tuke, M.P., 20 Charlotte Square,	1864
	Dr F. J. Turnbull, 6 Randolph Place,	1896
	Dr Dawson Turner, 37 George Square,	1890
	Dr Logan Turner, 27 Walker Street,	1892
	Dr Underhill, 8 Coates Crescent,	1872
255	Dr Henry Wade, 32 Alva Street,	1903
	Dr Norman Walker, 7 Manor Place,	1891
	Mr D. Wallace, C.M.G., 11 Rutland Street, *Secretary*,	1887
	Dr David Waterston, 15 Greenhill Gardens,	1900
	Dr Douglas Chalmers Watson, 22 Coates Crescent,	1894
260	Dr Charles Heron Watson, 16 Charlotte Square,	1903
	Sir Patrick Heron Watson, 16 Charlotte Square,	1856
	Dr A. D. Webster, Belleville Lodge, Blacket Avenue,	1883
	Professor David A. Welsh, M.D., Sydney, Australia,	1900
	Dr James Wilson, 53 Inverleith Row,	1900
265	J. L. Wilson, Esq., 4 Buccleuch Place,	1883
	Dr Russell E. Wood, 9 Darnaway Street,	1883
	Professor John Wyllie, 44 Charlotte Square,	1868
	Dr P. A. Young, 25 Manor Place,	1870

(*b*) Members Exempted under Rule V. from paying the Annual Subscription.

		Date of Admission.
	Dr D. Aitken, 152 *Packington Street, Islington, London, N.*,	1887
270	Dr D. H. Anderson, 131 *Abbey Road, Barrow-in-Furness*,	1887
	Dr Archibald, *Hazelden, Wimborne Road, Bournemouth*,	1882
	Dr J. A. Armitage, 58 *Waterloo Road, Wolverhampton*,	1887
	Dr W. Badger, *Penicuik*,	1882
	Dr Edwin Baily, *Oban*,	1883
275	Dr Andrew Balfour, *Portobello*,	1874
	Dr Andrew Balfour, *Khartoum*,	1895
	Dr J. H. Balfour, *Portobello*,	1881
	Dr Alexander Ballantyne, *Dalkeith*,	1872
	Dr Noel D. Bardswell, *Midhurst Sanitorium, Sussex*,	1901
280	Dr W. H. Barrett, 29 *Park Crescent, Hesketh Park, Southport*,	1890
	Dr George T. Beatson, 7 *Woodside Crescent, Glasgow*,	1895
	Surgeon-Captain C. H. Bedford, *Bengal Army, care of W. Watson & Co.*, 28 *Apollo Street, Bombay*,	1889
	Dr Charles Begg, 62 *Pulteney Street, Bath*,	1900
	Dr Benjamin D. C. Bell, *Kirkwall*,	1889
285	Dr G. J. H. Bell, *Bengal Army*,	1884
	Dr G. H. Bentley, *Kirkliston*,	1877
	Professor Richard J. A. Berry, M.D., *University of Melbourne*,	1893
	Dr J. S. Beveridge, *Melton Constable, Norfolk*,	1861
	Dr J. W. Black, 15 *Clarges Street, Piccadilly, London, W.*,	1865
290	Dr P. G. Borrowman, *Galvalmore, Crieff*,	1893
	Dr D. G. Braidwood, *Halkirk, Caithness*,	1889
	Dr David Brodie, *Singbsby Villa, Royal Park Road, Finchley, N.*,	1865
	Dr Andrew Brown, 1 *Bartholomew Road, Kentish Town, London, N.W.*,	1884
	Dr John Brown,	1878
295	Dr J. Macdonald Brown, 2 *Fragnall, Hampstead, London, N.W.*,	1883
	Dr William Brown, *Braemar*,	1904
	Dr Lewis C. Bruce, *The Asylum, Murthly, Perthshire*,	1895
	Dr William Hartley Bunting, 22 *Islington Row, Edgbaston, Birmingham*,	1900
	Dr D. A. Carruthers, 5 *Charlotte Street, Perth*,	1901
300	Dr S. W. Carruthers, 44 *Central Hill, Norwood, London, S.E.*,	1899
	Dr R. D. Clarkson, *Falkirk*,	1896
	Professor Cleland, *The University, Glasgow*,	1864
	Dr A. R. Coldstream, 24 *Lung Arno Nuovo, Florence, Italy*,	1878
	Dr William Craig, *Cowdenbeath, Fife*,	1894
305	Dr J. R. Crease, 2 *Ogle Terrace, South Shields*,	1885
	Dr J. Frank Crombie, *North Berwick*,	1897
	Dr Bampylde Daniell, *Junior Conservative Club, Albemarle Street, London, W.*,	1903
	Dr P. M. Deas, *Wonford House, Exeter*,	1868
	Dr A. Halliday Douglas, 12 *Dean Park Crescent*,	1842
310	Dr A. Home Douglas, 5 *Rue de Russie, Nice, France*,	1889
	Dr J. J. Douglas, 42 *Central Hill, Upper Norwood, London*,	1891
*	Dr Thomas Easton, 23 *East Park Terrace, Southampton*,	1894
	Dr J. W. Eastwood, 18 *Farndon Road, Oxford*,	1871
	Dr F. A. Elkins, *The Asylum, Leavesden, near King's Langley*,	1893
315	Dr R. W. Felkin, 48 *Westbourne Gardens, Bayswater. London, W.*,	1885
	Dr Samuel Fergusson, *Lylestone House, Alloa*,	1902
	Right Hon. Sir R. B. Finlay, Q.C., LL.D., *Middle Temple, London*,	1864

		Date of Admission.
	Dr Charles Forsyth, *Alexandra Buildings, Hong-Kong*,	1903
	Dr Simson C. Fowler, *Juniper Green*,	1892
320	Dr F. W. Foxcroft, 47 *Newhall Street, Birmingham*,	1893
	Dr Dyce Fraser, 163 *Cornwall Road, London, S.W.*,	1883
	Professor Gamgee, 8 *Avenue de la Garve, Lausanne, Switzerland*,	1863
	Dr William Gayton, 11 *Redbourne Avenue, North Finchley, London, N.*,	1886
	Dr T. Paul Gray, 82 *Belsize Park Gardens, Hampstead*,	1899
325	David M. Greig, Esq., 25 *Tay Street, Dundee*,	1892
	Dr W. C. Greig, *Otago, New Zealand*,	1884
	Dr George K. Grimmer, *Canada*,	1897
	Dr Groesbeck, *Cincinnati*,	1875
	Dr W. B. T. Gubbin, *Redland Grove, Bristol*,	1893
330	Dr John Haddon, *Denholm, Roxburghshire*,	1883
	Dr Archibald Hall, *Montreal*,	1853
	Professor D. J. Hamilton, *The University, Aberdeen*,	1876
	Dr J. W. Hamp, *Penn Road, Wolverhampton*,	1887
	Dr John Hardie,	1895
335	Dr A. W. Hare, *Newquay, Cornwall*,	1883
	Dr J. Home-Hay, *Alloa*,	1880
	Professor J. Berry Haycraft, *Llanishen, Cardiff*,	1889
	Dr Stanley Haynes, *Malvern, Worcestershire*,	1864
	Dr R. Dundas Helm, 13 *Portland Square, Carlisle*,	1892
340	Dr David Hepburn, Professor of Anatomy, *University College, Cardiff*,	1902
	Dr R. E. Horsley, *Crail, Fife*,	1886
	Dr James Hunter, *St Catherine's, Linlithgow*,	1890
	Dr W. Hunter, 103 *Harley Street, Cavendish Square, London, W.*,	1887
	Dr David Huskie, *Moffat*,	1899
345	Dr Robert Inch, *Gorebridge*,	1887
	Dr W. Wotherspoon Ireland, 1 *Victoria Terrace, Musselburgh*,	1883
	Dr D. W. Johnston, *P.O. Box* 2022, *Johannesburg, South Africa*,	1893
	Dr James Johnston, 53 *Princes Square, Bayswater, London, W.*,	1871
	Dr J. Carlyle Johnstone, *The Asylum, Melrose*,	1882
350	Dr J. Keay, *Bangour Asylum, Uphall*,	1887
	Dr A. J. Keiller, *New Brunswick, Canada*,	1889
	Dr George Keith, *Moidart Cottage, Currie*,	1845
	Dr Skene Keith, 58 *Upper Berkeley Street, Portman Square, London, W.*,	1885
	Dr W. Scott Lang,	1868
355	Dr J. W. Leech, 12 *Eldon Square, Newcastle-on-Tyne*,	1901
	Professor R. F. C. Leith, M.B., *The University, Birmingham*,	1886
	Dr John Linton, *Aberdour*,	1863
	Dr G. R. Livingston, 47 *Castle Street, Dumfries*,	1901
	Dr Donald Macaulay, *Jumpers' Gold Mining Co., P.O. Box* 1043, *Johannesburg*,	1895
360	Dr W. A. M'Cutchan, *Fulbourn Asylum, Cambridgeshire*,	1897
	Dr F. R. Macdonald, *Inveraray*,	1860
	Dr John A. Macdougall, *Cannes, France*,	1875
	Dr William C. M'Ewan, *Prestonpans*,	1892
	Professor Sir J. M'Fadyean, 101 *Great Russell Street, London, W.C.*,	1888
365	Dr F. Reid Mackay, *New Zealand*,	1899
	Dr John Mackay, *Aberfeldy*,	1881
	Dr W. B. Mackay, 23 *Castlegate, Berwick-on-Tweed*,	1893
	Professor M'Kendrick,	1870
	Dr James Mackenzie, 68 *Bank Parade, Burnley, Lancashire*,	1894

		Date of Admission.
370	Dr Robert Mackenzie, *Napier, Nairn*,	1893
	Dr William A. Mackintosh, 3 *Pork Terrace, Stirling*,	1896
	Dr David W. Maclagan, *care of Dr Harrison, Eltham, Taranaki, New Zealand*,	1901
	Dr J. Shaw M'Laren, *India*,	1887
	Dr Roderick M'Laren, 23 *Portland Square, Carlisle*,	1882
375	Dr William B. MacTier, 7 *Queen's Gardens, St Andrews*,	1901
	Dr Samuel Macvie, *Chirnside*,	1897
	Dr J. W. Martin, *Charterhall, Newbridge, Dumfries*,	1888
	Dr Andrew W. Messer, *Lemington, Scotswood-on-Tyne*,	1899
	Dr James Middlemass, *Boro' Asylum, Ryhope, Sunderland*,	1895
380	Dr E. A. Mills-Roberts, *Bethesda, Bangor*,	1897
	Dr J. Moolman, *Cape of Good Hope*,	1877
	Dr E. C. Moore,	1892
	Dr J. E. Moorhouse, 6 *Melville Terrace, Stirling*,	1901
	Dr A. E. Morison, *Wellington Road, West Hartlepool*,	1889
385	Dr J. Rutherford Morison, 14 *Saville Row. Newcastle-on-Tyne*,	1882
	Professor Robert Muir, *The University, Glasgow*,	1894
	Dr D. G. Macleod Munro, 1 *Oriel Villas, Cheltenham*,	1898
	Dr Peter Murray, *Durie Street, Leven*,	1904
	Dr Andrew Scott Myrtle, *Harrogate*,	1859
390	Dr Leith Napier, *The General Hospital, Adelaide. South Australia*,	1879
	Dr T. Goodall Nasmyth, *Cupar-Fife*,	1884
	Dr Ernest F. Neve, *Srinagar, Kashmir, India*,	1884
	Dr Peter Orphoot, *Greenhythe, North Berwick*,	1865
	Dr T. Wyld Pairman, *Hinemoa, Moffat*,	1884
395	Professor Bell Pettigrew, *St Andrews*,	1873
	Dr Alexander Peyer, *Zürich*,	1893
	Dr J. A. Philip, *Rue Victor Hugo, Boulogne-sur-Mer, France*,	1878
	Dr J. H. Pringle, 172 *Bath Street, Glasgow*,	1886
	Dr S. Hale Puckle, *Bishop's Castle*,	1885
400	Dr R. B. Purves, 5 *Minster Yard, Lincoln*,	1899
	Dr Rattray, *Portobello*,	1874
	Dr G. M. Robertson, *The Copse, Larbert*,	1901
	Dr J. Maxwell Ross, *St Ruth's, Maxwelltown, Dumfries*.	1882
	Dr J. R. Home Ross, *Burmah*,	1888
405	Dr Joseph C. Ross, *Withington*,	1884
	Dr S. Rumboll, *Cavendish House, Leeds*.	1887
	Dr Thomas Russell, 47 *Westmuir Street, Parkhead, Glasgow*,	1888
	Dr Gordon Sanders, *Cannes, France*,	1892
	Dr F. A. Saunders, *Grahamstown, South Africa*,	1884
410	Dr Thomas R. Scott, *Musselburgh*,	1884
	Dr W. Vernon Shaw, 42 *Yorkersgate, Malton, Yorks*,	1905
	Dr C. A. E. Sheaf, *Ovalu, Fiji*,	1871
	Dr Herbert D. Shepherd, *Greystone, Arnside, Westmorland*,	1903
	Dr G. F. Shiels, 229 *George Street, San Francisco*,	1887
415	Dr W. H. Shirreff, *Port Fairy, Victoria, Australia*,	1883
	Dr William Simmers, *Gordon, Berwickshire*,	1894
	Dr T. Skinner, 6 *York Place, Portman Square, London, W.*,	1856
	Dr John Smith, *Brycehall, Kirkcaldy*,	1889
	Dr William Smith, *America*,	1890
420	Dr W. Ramsay Smith, *The General Hospital, Adelaide, South Australia*,	1892
	Dr Van Someren, *Northwood, Middlesex*,	1845
	Dr A. M. Stalker, 140 *Nethergate, Dundee*,	1893
	Dr Graham Steell, 96 *Moseley Street, Manchester*,	1877
	Professor Stephenson, *University, Aberdeen*,	1861
425	Dr John Stevenson,	1900
	Dr Robert Stirling, 4 *Atholl Place, Perth*,	1891

		Date of Admission.
	Professor Ralph Stockman, *The University, Glasgow*, .	1891
	Dr H. R. Storer, *Newport, Rhode Island, U.S.*, . .	1855
	Dr John Struthers, *Ngamakwe, Transkei, South Africa*, .	1895
430	Dr J. F. Sturrock, *Arima, Broughty Ferry*, . . .	1887
	Professor J. Symington, *Queen's College, Belfast*, . .	1878
	Dr C. C. Teacher, *North Berwick*, ...	1887
	Dr C. Templeman, *Sanitary Office, Bell Street, Dundee*, .	1891
	Dr J. A. Douglas Thompson, *Comberton House, Halesowen, Manchester*, ...	1901
435	Dr J. Stitt Thomson, *Castle Hill House, Lincoln*, . .	1877
	Dr J. Batty Tuke, Jr., 20 *Charlotte Square*, . . .	1886
	Dr R. S. Turner, *Keith*, ...	1867
	Dr T. Edgar Underhill, *Dunedin, Burnt Green, Worcestershire*, ...	1885
	Dr A. R. Urquhart, *Murray House, Perth*, . . .	1893
440	Dr Gopal Govind Vatve, *care of H.H. the Rajah of Miraj, Bombay, India*, ...	1895
	Dr Walter de Watteville, *Kingussie*, ...	1902
	Dr A. B. Whitton, *Aberchirder*, . . .	1886
	Dr George R. Wilson, *Mavisbank, Polton*, . . .	1892
	Dr H. Douglas Wilson, 3 *Rippon Road, Harrogate*, . .	1904
445	Professor G. Sims Woodhead, *Pathological Laboratory, New Museums, The University, Cambridge*, . . .	1883
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TRANSACTIONS

OF THE

MEDICO-CHIRURGICAL SOCIETY OF EDINBURGH

FOR SESSION LXXXIV., 1905-6

Meeting I.—November 1st, 1905

PROFESSOR JOHN CHIENE, C.B., *President, in the Chair*

I. ELECTION OF OFFICE-BEARERS

The following gentlemen were elected office-bearers:—*President*, James O. Affleck, M.D., F.R.C.S., Edin., F.R.C.P., Edin.; *Vice-Presidents*, Professor Sir John Halliday Croom, Professor William S. Greenfield, and Dr. Byrom Bramwell; *Council*, Dr John Macpherson, Mr Alexander Miles, Dr J. Crawford Dunlop, Dr William Elder, Professor John Chiene, C.B., Mr Alexis Thomson, Dr R. A. Fleming, and Dr F. D. Boyd; *Treasurer*, Dr H. Harvey Littlejohn; *Secretaries*, Dr G. Lovell Gulland and Mr David Wallace, C.M.G.; *Editor of Transactions*, Dr William Craig.

II. ELECTION OF MEMBERS

Henry Grey Brown, M.D., C.M., 1 Cluny Avenue; John Herbert Gibbs, F.R.C.S., Edin., L.D.S., 7 Coates Place; J. H. Montgomery Bell, M.B., Ch.B., 200 Bruntsfield Place; and W. Morrison Milne, M.B., F.R.C.S., Edin., 11 Newington Road, were elected Ordinary Members of the Society.

III. Treasurer's Annual Statement

EDINBURGH MEDICO-CHIRURGICAL SOCIETY'S ACCOUNTS

FOR SESSION 1904-1905.

INCOME				EXPENDITURE			
			£160 16 2				£141 14 0
Entrance Fees	£10 9 6			Turnbull & Spears, printing Vol. XXIII. of Transactions, £79, 1s. 7d. Sundry printing, £1, 16s. 3d.	£80 17 10		
Annual Subscriptions (with arrears £2, 10s., and prepaid £1)	127 10 0			Neill & Co., general printing	25 13 9		
Subscriptions for Transactions	2 15 0			Frazer, Microscopes and Lantern	6 14 0		
Sale of Transactions	3 4 8			Padon & Co., Stationery	6 7 0		
Interest on Caledonian Railway Stock	13 6 0			M'Callum, Tea and Coffee	6 3 8		
Interest on Deposit Receipts	3 11 0			Rent of Hall	10 0 0		
	£160 16 2			Hallkeeper	1 6 0		
				Collector	3 10 0		
				Petty Cash	1 1 9		
					£141 14 0		
Balance in Bank at October 18, 1904			28 11 9	Balance in Bank at October 27, 1905			47 13 11
			£189 7 11				£189 7 11

STATE OF FUNDS.

At November 1904.				At November 1905.			
On Deposit Receipt			£260 0 0	On Deposit Receipt			£260 0 0
Caledonian Railway Debenture Stock			484 11 6	Caledonian Railway Debenture Stock			484 11 6
Balance in Bank			28 11 9	Balance in Bank			47 13 11
			£773 3 3				£792 5 5

HARVEY LITTLEJOHN, *Treasurer.*

We have examined the Accounts and Vouchers and find them correct.

GEORGE HUNTER, M.D.,
HARRY RAINY, M.D., } *Auditors.*

November 13th, 1905.

IV. Exhibition of Patients

1. *Mr H. J. Stiles* exhibited—

(*a*) A girl, aged 8 years, five months after operation for GENERAL PERITONITIS, following gangrenous appendicitis. The abdomen was opened in the right iliac fossa three days after the onset of the attack ; the gangrenous appendix, which ascended in front of the kidney, was removed. The peritoneal cavity everywhere contained an abundance of thick pus. No attempt was made to swab out or wash out the abdomen, but free drainage was provided by introducing a rubber tube through openings made in the following situations, namely, the right costal margin, in the middle line, immediately above the umbilicus, from each iliac fossa through the corresponding loin, and from the middle line below the umbilicus through the posterior fornix of the vagina. After introducing the tubes they were rapidly syringed through with saline to make sure that they were clear. The tubes were removed and replaced on the fourth day ; subsequently they were shortened and smaller ones substituted. The vaginal tube was removed on the eighth day ; by the fourteenth day all the tubes were removed. The patient left the hospital in good health during the seventh week. Mr Stiles remarked that he had had better results in general peritonitis since he paid more attention to drainage and less to washing out. Drainage openings could be made very rapidly, and provided they were only just large enough to admit a good-sized tube, there was no risk of subsequent hernia.

(*b*) A patient nine months after operation for UNUNITED FRACTURE OF THE FEMUR. The patient, a male aged 31, was admitted into Chalmers Hospital three and a half months after the injury. While he and his brother were engaged in blasting operations the charge accidentally exploded, with the result that both fell to the bottom of the quarry, a distance of forty feet. The brother lost an eye and sustained a compound fracture of the elbow.

When the patient now exhibited was admitted into hospital, it was found that the thigh could be freely moved in all directions at the seat of fracture, which was a little below the junction of the middle and lower thirds of the femur. There was marked shortening, amounting to $2\frac{1}{2}$ inches. The

lower end of the upper fragment could be distinctly felt anteriorly a finger's breadth above the upper border of the patella; the upper end of the lower fragment was situated posteriorly and externally, a little above the upper angle of the popliteal space. By no amount of weight extension could the shortening be reduced.

Operation.—With the patient placed in the prone position, a 6-inch incision was carried from the head of the fibula upwards in the interval between the biceps and the vastus externus. The clearing of the ends of the fragments was a very laborious business, due to the density of the cicatricial tissue. As the large vessels could not be identified, it was considered safer to work without a tourniquet. Before the ends could be brought into proper apposition, it was found necessary to remove an inch and a quarter from the upper and one inch from the lower fragment. A Gigli's saw was used to make the sections. For the purpose of fixation an aluminium plate and eight screws were used, four being introduced into each fragment. The wound healed *per primam*, and the patient left hospital during the eighth week with the limb in plaster. He was soon able to dispense with crutches, and for some weeks he has been doing a full day's work. He had no trouble with the limb until a week or so ago, when he began to complain of slight pain in the scar, one part of which had become stretched, and under this portion there was a small collection of serum. The patient was readmitted into hospital, and on opening up the scar a granulation-tissue track was traced to one of the screws, which had become slightly moveable. Although the plate was firmly fixed, it was deemed advisable to remove it, so as to make sure that there would be no further trouble. This turned out to be a matter of considerable difficulty on account of the firm fixation of the plate. There was very firm union without extensive callus.

(*c*) Patient, aged 39, after operation for TORSION AND STRANGULATION OF A SARCOMATOUS ABDOMINAL TESTICLE. The patient had no trouble until seven months ago, when he was suddenly seized while sitting at his work with severe abdominal pain and vomiting. The pain lasted for a day, and in three days he felt quite well again. In the four subsequent attacks which the patient has had the severe pain lasted longer, and was attended with some increased frequency of micturition.

Mr Stiles saw the patient just before the last attack and diagnosed torsion of the abdominal testicle. An exploratory operation was advised with the view of removing the testicle. An oblique incision in the right iliac fossa revealed a smooth, oval, hard, plum-coloured swelling, the size of a large lemon. It was found that the tumour was pretty firmly jammed in the true pelvis. After enlarging the incision and delivering the tumour out of the pelvis, it was found to possess a twisted pedicle the thickness of the thumb. The twist, which was directed inwards towards the middle line, was made up of two and a half turns. The appearance of the tumour, the pedicle, and the character of the torsion bore a striking resemblance to a twisted ovarian tumour. The pedicle was transfixed, ligatured with strong catgut, and the tumour removed. To the naked eye the bisected tumour suggested a diffused hæmorrhagic infarction, the result of strangulation ; microscopic examination, however, showed that we had to deal with a hæmorrhagic extravasation into a round cell sarcoma. There were no enlarged glands, nor was there any evidence that the disease had extended along the pedicle. It is now three months since the operation, and so far there is no suspicion of recurrence.

(*d*) A girl, aged 6 years, showing an aggravated degree of Volkmann's ISCHÆMIC CONTRACTURE following a fracture of the lower end of the humerus thirteen weeks previously. Mr Stiles reminded the Society that in December of last year Mr Caird had exhibited two cases of this condition. A fortnight ago Mr Rowland published an interesting paper on the condition in the *Lancet* for October 21st. The deformity shown in the patient before the Society was precisely the same as that so well illustrated in Mr Rowland's article.

The patient was sent to the Children's Hospital as an atypical case of musculo-spiral paralysis. It was found, however, that the main feature of the condition was the great firmness, rigidity, and contracture, more especially of the flexors of the fingers, both superficial and deep. It was quite impossible to straighten the fingers with the wrist in the natural position, and even when the latter was forcibly flexed the fingers could not be completely straightened. There was complete inability to flex the fingers voluntarily ; there was, however, very slight voluntary movement in the extensor of the

fingers and in the intrinsic muscles of the hand. There was almost complete anæsthesia of the skin over the whole hand, and on the back of the fingers were three superficial ulcers. The patient was seen almost immediately after the accident, when the limb was put up in the fully flexed position with cotton wool and a bandage. The following day a poroplastic splint, extending from the shoulder to the tips of the fingers, was applied. When this apparatus was removed three days after the injury, very extensive swelling was revealed in the region of the elbow, and extending down the forearm as far as the hand. A week later the splint was again removed and passive movement begun. This was continued regularly during the next three weeks. No doubt the extensive effusion which had taken place after the splints had been applied had interfered with the circulation to such an extent as to seriously affect the nutrition of the muscles, and thus to bring about the fibrosis and contraction. Mr Stiles remarked that he intended to follow the plan adopted by Mr Raymond Johnson, namely, to excise a portion of the radius and ulna so as to enable the fingers to be straightened out and maintained in the extended position by means of splints. The opportunity would be taken to remove small portions of the degenerated muscle for microscopic examination.

V. Exhibition of Specimens

1. *Dr T. Shennan* exhibited two microscopical preparations of the Spirochæte pallida (Schaudinn and Hoffmann), which is met constantly in primary and secondary syphilitic lesions. A large body of evidence has been adduced to support its etiological relationship to that disease.

One preparation showed the Spirochæte pallida and the Spirochæte refringens in the same field. This was from a hard chancre. The other slide showed the Spirochæte pallida in the bloody fluid squeezed out from a papular Syphilide.

2. *Mr Alexander Miles* exhibited—

(*a*) Seven prostate glands, recently removed by Suprapubic operation.

(*b*) Stomach removed by Gastrectomy for Carcinoma. Recovery.

(*c*) Two specimens of ruptured extra-uterine gestation, removed by operation for hæmorrhage.

3. *Mr H. J. Stiles* exhibited—

(*a*) Tuberculous kidney.

(*b*) Calculus pyonephrosis.

(*c*) Hydronephrosis with hypertrophy of opposite kidney.

(*d*) Gangrenous gall-bladder with perforation.

(*e*) Strangulated intra-abdominal testicle, removed by laparotomy.

(*f*) Prostate, removed by Freyer's method.

(*g*) Portion of small intestine resected for fæcal fistula following suppurative appendicitis.

(*h*) Malignant stricture of œsophagus.

(*i*) Carcinoma of ascending colon.

(*j*) Fæcal mass impacted above malignant stricture of pelvic colon.

(*k*) Carcinoma of rectum, *three specimens*.

VI. Original Communications

1. THE MEDICO-CHIRURGICAL SOCIETY OF EDINBURGH FIFTY YEARS AGO

VALEDICTORY ADDRESS

By Professor John Chiene, C.B., M.D., F.R.C.S.Ed., Retiring President

Gentlemen,—My term of office has come to an end, and I have now the pleasing duty to thank you for your invariable courtesy to me personally, and to say a few words of the nature of a *Vale* or farewell as your President.

Looking back on past addresses by my Presidential forebears, the majority have dealt with the work of the Society during the term of Presidential office of the speaker.

This method has never appealed to me. "No age can write its own history." The work recently done by this Society cannot be correctly judged by me, at any rate. It is too recent. I am too near to it.

Fortunately I have heard, as an example for me to follow, valedictory addresses from your Presidents taking a more distant view, sometimes looking back, sometimes looking for-

ward. I avoid the latter course; it is too dangerous, and I for a moment would like to look back in the annals of the Society.

This is a year of centenary celebrations: John Knox, Sir Thomas Browne, Trafalgar, the Royal College of Surgeons of Edinburgh, and the Royal Medico-Chirurgical Society of London.

All are interesting. Every true Scot is a theologian; it is in his blood. We don't object to a fight. As members of the medical profession the quater-centenary of our College and the tercentenary of Sir Thomas Browne specially appeal to us. I am tempted to-night to linger over Sir Thomas Browne. I cannot begin, because I would find a difficulty in ending.

A cotemporary of Wm. Harvey and Izaak Walton, I always think of these three men together; and the great lesson, of all importance in this age of hurry and putting other people right, that they seemed to me to teach is, the value of time for contemplation and tolerance. But I pass them all by; and although our Society cannot yet hold its centenary, I can, to-night, ask you to look back for fifty years. I think I am tempted to this date when I look at the names of the men who were then the active members in this Society. I find men whom I looked up to in the early sixties as my teachers, and if there is anything that seems to me to appeal to the best in us, it is to place on record our thanks to those who have influenced us in our youth. An old favourite of mine, Marcus Aurelius Antoninus, Imperator et Philosophus, begins his simple story of his life with such a recital. One of his axioms is, "a man should always have in his mind some one or other of the ancient worthies." In 1855, some of these men, of whom I wish to say a few words, were pointed out to me by my seniors, when I was an Academy schoolboy, as worthy citizens of no mean city. Then, as now, there were the successful men worked off their legs, seeing this one and that one; and there were the quiet, easy-going men, who, perhaps, spent a happier life, at any rate a more restful life. Both had their duties and their responsibilities: "the universe has need of all men," or, as the old Scottish lady said, "it taks aw kinds o' buddies to mak a world."

In 1855, Dr William Sellar was your President. I remember him living in Northumberland Street, and even when

I was a boy he appealed to me. I knew him well by sight. All men spoke well of him. There was something kindly, Pickwickian, in his appearance. I never heard that he had much practice. I think he had boarders, possibly apprentices, who attended the medical classes in the College. I would imagine that they had an easy time. It was about this time that the profession entertained him to a banquet in the Granton Hotel. These were the old ferry days, when Granton was an important port, and when the Granton Hotel was as the Lord Warden in Dover. Nowadays that route to Fife is in its first tunnel part given up to the growth of mushrooms. From Scotland Street to the sea there is now only mineral traffic!

Of the Vice-Presidents I gladly recall my first teacher in Surgery, the late Professor Miller, a polished, courteous gentleman, whose honesty of character, and constant desire to do the right thing, endeared him to his students.

Amongst the Council I note H. D. Littlejohn, *semper juvenis*. He was at that time the editor of the *Edinburgh Medical Journal*, and I thank him for the fulness of the reports of the Medico-Chirurgical Society. His work fifty years ago has made my work easy to-night. He never thought when he took care to have good reports of the meetings that the President of 1905 would directly benefit from his devotion to duty fifty years ago. Good work, whenever and wherever done, will always bear fruit sooner or later. He began his duties as editor in July 1855, and in the second number he very well illustrates the spirit in which he worked by the kindly support given to the Medical Benevolent Fund. He says: "In this an early number of the *Edinburgh Medical Journal* we feel a peculiar pleasure and recognise a singular, although almost accidental, fitness and propriety in noticing the operations of the Medical Benevolent Fund." "The medical profession is essentially a benevolent one, and we hesitate not to assert the almost unbounded scope to which extends the private and unheard of charity of its members;" and he very truly points out "that the object of this fund is to obtain, to systematise, and to facilitate the benevolent action of the profession towards itself," and he speaks of "the active benevolence of the profession towards its own members as a solemn duty incumbent on it."

It will always be well with our profession when such are its guiding principles.

On the Council I note a man who, as a teacher of Pharmacy and Minor Surgery, was a well-known figure in my student days, Dr David Gordon, whom we irreverently called "Longus Colle." He, I remember, had a special pair of tooth forceps which could with great advantage be applied to every variety of tooth.

Another member of Council, Daniel Rutherfurd Haldane, was my teacher in Systematic Medicine in 1864. He was in 1855 Pathologist to the Royal Infirmary and Lecturer on Pathology in the Extra-Academical School. I look back to his lectures as the most polished, concise, and practical course of instruction in Medicine—in the wide sense of the term—to which I ever listened. He had that all-important faculty in every teacher of being able to apportion out his subject with a precision and due sense of the proper proportion of every branch of the subject, so that without any hurry at the end of the session we each felt that Dr Haldane had made the very most of the time at his disposal.

Dr Alexander Wood was another member of the Council. I knew him personally, and he often said an encouraging word to me when I was a student. I had no experience of him as a teacher, but he will always be remembered as the man who introduced the hypodermic syringe to the notice of the profession.

It was in this year that the famous struggle for the Chair of Practice of Physic took place. It was a triangular duel—Bennett, Laycock, and Wood; and Wood ran Dr Laycock a very close second—Laycock 17, Wood 15, votes in the Town Council, which then had the patronage in its hands.

The Treasurer of the Society was Dr John Struthers, then an extra-academical lecturer on Anatomy. Two years before he had taken Prof. Goodsir's class during his absence from illness. He afterwards became Professor of Anatomy in Aberdeen, and after his retirement came back to Edinburgh to take an active part as a manager in the Leith Hospital and Edinburgh Royal Infirmary, and to fill with energy the Presidentship of the Royal College of Surgeons. All his life he lived fully, and in the records of the Medico-Chirurgical Society there was no more active member than John Struthers.

The Secretaries of the Society were Dr W. T. Gairdner and Dr Warburton Begbie, who, along with Struthers, took a most active part in the meetings of the Society.

Of Dr Gairdner, now Sir William Gairdner, who is still with us, and acutely interested in everything connected with the profession of which he is an ornament, it may truly be said that from the day on which he became a physician in the Royal Infirmary until he retired from the Chair of Medicine in Glasgow, he was always in the forefront, and, in my opinion, the greatest physician in Scotland.

He was also, when in Edinburgh, like Dr Sellar, an inhabitant of Northumberland Street, and a personal friend of my father's. I remember well the first day I spoke to him in the dining-room at No. 27, my father's house. Then an Academy boy, it must just have been 50 years ago. I had some trifling ailment, I forget what, and he came to see me and advised a rest from school for a time. I fully appreciated the kindliness of his prescription. From that day I have always felt a respect and love for Sir Wm. Gairdner.

In 1855, medical politics were burning questions, and I was early taught the fact that Dr Gairdner in those days was a reformer who did not always see eye to eye with the Professoriate—he was one of the "outs," and took a friendly interest in the "ins," which did not always meet with their approval. The two forces must always be at work as long as the world lasts—those who desire to change the steam power, those who act the part of the Westinghouse brake and think things are very well as they are, and that great care must be taken to proceed cautiously. A very distinguished professor travelled one day to Fife with my father, and as they left Burntisland he pointed to an unsigned article in the *Scotsman* and said with a shrug of his shoulders, "the work of Willie Gairdner." That day at dinner my father told me the story. "No age can write its own history" is specially applicable to Sir W. T. Gairdner. We don't really yet see the inherent greatness of the man, the very nearness of the subject renders a just estimate impossible. You cannot estimate the height of a hill by standing in the near valley and seeing only the top of the first knoll; even if you climb to that point you only see another rise—the summit is not yet.

Of Warburton Begbie, the dear friend of many of us,

whose short meteoric career is well known to all who lived before 1876, I will find words with difficulty to be your efficient mouthpiece on this occasion.

I think when I looked up the minutes of 1855, and found Warburton Begbie was the Junior Secretary, it appeared that, in the language of the philosophers, "now all these points could never have been compassed without a protection from above, and the Gods presiding over fate"—in other words, I felt that I was a fortunate man to be permitted to put on record to the present generation a few aspects of Warburton Begbie's character, recalling, if possible, what he was to many that knew him, and telling the new generation of that physician who, after he served as a physician in the Infirmary from 1855 to 1865 (the year I graduated), and more especially after his father's death in 1869, until his own too early death in 1876, was the man whom everybody must see if he was seriously ill. It was said, in a county to which he so often went, "Begbie has been sent for," and the friends of the patient knew that all had been done that could be done.

He was killed by his work and worries; he was worn out at 50.

What lesson did Begbie teach his pupils? The value of sympathy, courtesy, and concentration. He was ever ready—however tired—to help anyone in suffering; his professional brethren and his students went to him in all their troubles; how gentle and kind he was. He taught us another lesson—to believe in your power of being able to help, however apparently hopeless the case, and he also taught us to do what lay immediately to our hand with all our might. There was no "impatience of attention" in Warburton Begbie. He never checked the patient telling his own story in his own way. He never jumped in and said he knew until he had heard all, and then he spoke with confidence. What a blessing he was to the friends of the patient—a word from Begbie often gave more comfort than a homily from another.

He had, above anyone I ever met, the faculty of impressing each of his patients—no matter what his status in life—with the feeling that his case absorbed all his interest.

Begbie's life seemed a long one—he did so much in the years. He only got into the thick of it, so to speak, after he left the hospital. I never remember him being away a day.

He was always among his patients. He generally spent three hours in the hospital—12 to 3—and then crossed Infirmary Street to his lecture on Practice of Physic. There was nothing of the drill sergeant about him—he ruled by love. He was what the Americans call a lovely man. He possessed three great faculties—a deep reverence for the past, a profound knowledge of the present, and an intense belief in the future. He had a wonderful combination of qualities, all of which went to making him the beloved physician.

I am indeed fortunate in bringing James Warburton Begbie before the junior members of the Medico-Chirurgical Society.

The year 1855 should be remembered in this school. It was the year in which Warburton Begbie, Joseph Lister, and Henry Littlejohn began their teaching career in Edinburgh as extra-academical teachers. The extra-academical school has been the nursery of many men who have made their mark in our profession. May it ever prosper. I gladly place on record how much I owe to it. It was my foster-mother when I needed help, and I trust I have never forgotten a remark made to me by an old extra-mural friend who lectured with me in the old Nicolson Square class-room when I was made Professor. "See that you don't kick away the ladder by which you have climbed." The ladder rests on the building into which I have climbed—in other words, both are essential.

The welfare of the extra-academical school is intimately bound up with the welfare of the University, and the welfare of the University on its medical side is bound up in a prosperous extra-academical school.

This is perhaps not the place, nor is this the time, to speak of the medical school, but as I see teachers before me and many who will be teachers, I cannot but tell you my opinion that we must bind the two elements more closely together. We have seen within recent years many new centres of medical education arising in the old country and in the empire beyond the seas. We cannot hold our position on the old lines, we must reorganise our forces—"What was good enough for twenty years ago is not good enough for to-day."

More closely bound together, more especially in the hospital, we will certainly be a stronger organic whole and

more able to contend in all friendliness with our opponents who are our own offspring. Let us avoid if possible the fate of the mother scorpion of Brazil. We have to see that, after bringing forth a brood of young scorpions, we are not eaten by our own progeny; or, as Sir Thomas Browne puts it, "I perceive a man may be buried alive and behold his grave in his own issue." For "man" read "University."

There is no necessity to go systematically over the work done during Dr Sellar's presidency. Let me only indicate the nature of the work. We find Professor Christison upholding the rights and privileges of the medical witness, and pointing out that unless some protection against the attacks of counsel is given that it will be difficult to get medical men to come forward.

He also asks what is your duty when you suspect a case of chronic poisoning; his verdict is, tell the victim of your suspicions, not the civil magistrate, nor the suspected person.

Mr Spence read a most interesting paper on a case of Hernia, in which strangulation was due to an irregular obturator artery. At that meeting the preparation of a similar case in which the artery encircled the neck of the sac and was not wounded—the operation having been performed by the late Dr R. T. Mackenzie—was shown by Dr Struthers. Dr Mackenzie's death had recently occurred in the Crimea, where he had gone to the seat of war as a volunteer.

Mr Spence's classical paper on Cario-necrosis was read before the Society at this time.

Dr Wm. Broadbent, when he was Professor Syme's house-surgeon, I think, read a paper on, and showed a case of, Tubercular Leprosy (or elephantiasis Græcorum) occurring in a patient from Stornoway, which elicited an interesting discussion, in which it was stated that leprosy was met with in Austria, and Dr Simpson (afterwards Sir J. Y. Simpson) stated that he had the day before seen a leper in his consulting-room, and he asked the question if the disease was returning again to this country? It was pointed out that the Swedes spoke of prevention by the "Scottish method," namely, castration of the males suffering from the disease. Dr Simpson hoped our Swedish friends would understand that this method of dealing with the disease was not now advocated in Scotland.

Then we have Dr Warburton Begbie on Rheumatism and

Chorea, a subject in which he took a deep interest. I remember well his interest in such cases when I was a clerk in his wards in 1864.

Dr Keith read a paper on "Manual Strangulation," which, knowing the doctor only as an authority on Midwifery, I took to be a paper dealing with some obstetrical condition, but it was an interesting medico-legal contribution—a subject which Dr Keith taught before he became a teacher of Midwifery.

There are also two cases of death under the administration of chloroform, one on the operating-table in the hands of Dr Mackenzie of Kelso, and the other in the dentist's operating-chair, a patient of Dr Nesbit's, and an interesting discussion arose as to the cause of death.

Dr Benjamin Bell has a paper on "Dilution as a principle of Therapeutics." It is in my opinion a most interesting paper. He presses for small doses in a high state of dilution; in fact he tries to imitate Nature in the Chalybeate springs and in natural purgative mineral waters.

The hæmorrhagic diathesis, excision and examination of the knee-joint, chylous urine, operation for cancer of cervix, all occupied the attention of the Society; and Dr Alison, Emeritus-Professor of Practice of Physic, had a paper on "Bleeding and its Values," apparently one of the last struggles of the decay of faith in the value of bleeding in all cases of acute inflammation. Dr Bennet did not attend the meeting at which the paper was read. It was the play of Hamlet without Hamlet. The discussion hinged on the change of type in disease, and that as the eruptive fevers varied in severity in different epidemics, so possibly there was a cycle of severity in inflammatory conditions.

Lister's first paper in the Medico-Chirurgical Society was read at this time. It was on a remarkable case of Hydrocele. Even in those early days Mr Spence was hypercritical of Mr Lister's views. Then, as now, interesting cases and interesting specimens were shown. Very often the meetings were entirely devoted to the exhibition of clinical and pathological material.

Occasionally a paper of general interest relieves the purely professional work of the Society, such as that by Dr Campbell Stewart of New York on "The Medical Schools in America." We learn that then, as now, all forms of illegitimate practice

were rampant in America, illustrating the great law that the greater the crowd, the less is the mental stability of the individual in the crowd.

I have said enough to show that our predecessors were active and energetic, desirous to learn, keen in argument, and liked above all things a thorough thrashing out of any subject at all costs. Perhaps nowadays we don't say all we think in the desire to be all things to all men. Perhaps it is well not to say all we think, but surely we can hold our tongues.

I notice in the Society a marked change ever since I was a member. We hear no longer of certain schools which pin their faith to one man ; a Simpson man, a Syme man, a Christison man no longer exist. We are more prepared to see good in everything, and hence good in everybody. I think there must be a preponderance of good in such an attitude towards our neighbour.

Three days ago I happened to be reading Emerson's paper on "Thoreau." (Thoreau, fortunately, is pretty well known to most men, but if it is a new name to anyone present, may I say, don't hesitate to make his acquaintance right speedily.) Thoreau was perhaps too critical, but there is much general truth in this sentence : "Not a particle of respect had he for the opinions of any man or body of men, but homage solely to truth itself" ; and as he discovered everywhere among doctors (all sorts of doctors) some leaning towards courtesy, it discredited them in his eyes, a frame of mind not altogether good, because apt to be intolerant. At the same time, if associated with that spirit which "will have no disciples"—in other words, neither giving nor asking a blind belief—then possibly most good will arise in discussion in medical societies and otherwise.

My time is more than up.

What are the lessons of the work of this Society fifty years ago ?

The world knows, or thinks it knows, more now than then.

A temporal bone was shown from the post-mortem room from a case of middle-ear disease with an abscess in the temporo-sphenoidal fossa ; they had no idea of opening the abscess.

Photography was first introduced in this year to the notice of the members.

The double stethoscope was shown for the first time on the same day.

Is a condyloma a specific disease, or is it a manifestation of the disease called syphilis?

A cleft palate was shown in which the treatment was a small eschar at the angle of the cleft, resulting in an ulcer which contracted as it healed, the process being repeated several times.

Things were then said which are now firmly believed, but which, said then, fell on barren ground and bore no fruit. Dr M'Cormac of Belfast communicated a paper on Tubercle to the Society on May 20, 1856, to the effect that tubercle is a vice of respiration, that the respiration of a vitiated atmosphere supplies all the conditions necessary for the development of tubercle, and he puts on record these words: "The problem to be solved was how best to secure, by day and night, air as pure as the great body of the atmosphere." Here in a sentence we have the open-air treatment of the present day. No one supported his view; in fact, it is the only paper which received no compliments. Kindly words were spoken of all the others; this only drew forth a warning. Things are being said now which fall on barren ground; the crowd is not yet ready for them.

I have enjoyed this desultory dive into the past, and perchance I have recalled to some of my hearers and compeers our early days when our world was young. For the other and major portion of my audience I can only say that I know nothing as a recreation or a solace in the stress of work—as on them the stress must now fall—better than a little time given to the contemplation of the ancient worthies. "There are three things the wise man ought to reverence: the laws of heaven, great men, and the words of good men." When Confucius penned these words he doubtless was thinking of his ancestors. As in the East, we find that ancestor worship was a most important factor in bringing to a successful issue the recent great struggle, happily now ended; so possibly in the battle of life in which nowadays the struggle is a severe one, a little ancestor worship may be a helpful thing.

Such a retrospect teaches us another lesson, that our time here must come to an end; some in early life, some in old age, some apparently when they can least be spared, but all at

the best and most proper time, we may be sure of that. We have all lost dear friends in this Society since I took the chair. We each sympathise with the other ; and for the near relations of those who have crossed the river, I am sure your heartfelt sympathy will be freely given.

I now hand over the pleasures and responsibilities of my office to my successor, in the full assurance that the best interests of the Medico-Chirurgical Society will be in safe keeping.

2. SOME OBSERVATIONS ON THE USE OF THE OPSONIC INDEX OF THE BLOOD IN THE DIAGNOSIS AND TREATMENT OF PULMONARY TUBERCULOSIS

By David Lawson, M.A. St And., M.D. Edin., F.R.S.E., Senior Physician to the Sanatorium at Banchory, and Ian Struthers Stewart, M.D. Edin., Clinical Pathologist to the Sanatorium at Banchory

Mr President and Gentlemen, — As there are no doubt some who may not have found it necessary to make themselves acquainted with the brilliant researches of Ehrlich abroad, and the more recent and hardly less brilliant work done more recently in this country by Wright, Douglas, Bulloch, and others on the subject of immunity, it has been considered desirable to preface the consideration of the subject-matter proper of this paper by an introduction of an explanatory nature. And in doing so, it is convenient to ask the question, "What is an Opsonin, and what is the part which it plays in the process with which we are dealing?" To that it may be replied that the word Opsonin is the term given to that substance or variety of substances which, circulating in the blood serum, facilitates the process of phagocytoses, and does so not by stimulating the phagocytes to further activity, but by lending to the bacilli an attractiveness which renders them welcome and acceptable to the white cells, which forthwith proceed to prey upon them in countless numbers. The term Opsonic Index corresponds to the numerical expression of the relative amount of Opsonins present in a person's blood, when compared with the amount contained in the blood of a healthy person. The meaning of this definition will, it is hoped, become clearer when we

have briefly detailed the process by which the Opsonin Index of a patient's blood is determined.

Let us assume that we have three small tubes containing respectively a small quantity of (*a*) healthy blood, (*b*) of healthy blood plus sodium citrate solution, and (*c*) an aqueous solution of tubercle bacilli. Tube (*a*) containing healthy blood is placed in an incubator at a temperature of 20° C., and maintained there for a period of twenty minutes. The effect of this is to facilitate clotting. It is then removed, and its contents centrifugalised. In this way complete separation of clot from serum is obtained, and the serum becomes readily available for purposes of investigation. Tube (*b*) is next dealt with. The effect of the presence of the sodium citrate solution is to prevent clotting. By centrifugalising we obtain in this case not two layers but three layers. The higher specific gravity of the red blood corpuscles results in their being driven to the bottom of the tube; the same factor determines the white corpuscles in taking up an intermediate position, and the serum is found to be present at the top as before. The serum is in this case pipetted off and destroyed, and the *white corpuscles* then become easily accessible. By centrifugalising the bacillary solution in glass (*c*) the coarser elements are driven to the bottom, where they appear as sediment, and the supernatent *fine aqueous solution* of tubercle bacilli supplies us with the third element, which is necessary for our investigation.

We then draw into a fine capillary tube equal quantities of these substances, namely, healthy serum, healthy white blood corpuscles, and bacillary solution. These are thoroughly mixed together, placed in an incubator for twenty minutes, and then withdrawn. The remainder of the process consists in making a film, and staining by the ordinary Ziel Neillsson process. It then becomes an easy matter to count under the high power of any ordinary microscope the number of bacilli which are found to be present in thirty consecutive multinuclear cells. In this way we obtain the first figure required for our determination. In this case assume that it is 30. The second figure is obtained in exactly the same way, with this difference only, that instead of using, as in tube (*a*), the blood of a healthy person, we use in tube (*d*) blood drawn from the patient whose index we are investigating. In this

way we secure a second figure. Assuming in this case that it is 15, by employing the figure obtained from the examination of healthy blood as our denominator, and that obtained from the examination of the patient's blood as numerator, we get a fraction—in this instance, 15 upon 30—which, being expressed in decimals, is 0.5. We would then say that the Opsonic Index of this patient's blood is 0.5. The whole process occupies about an hour and fifteen minutes to perform.

The course of research which we have undertaken is one which cannot be completely carried out under a period of three years. It must, therefore, be understood that the present paper, covering, as it does, only a small area of the field to be explored, is of an essentially preliminary nature. In this communication we submit for your consideration certain questions which have suggested themselves to our minds, and the means which we have taken to elicit answers to these. The cases of pulmonary tuberculosis studied—with the exceptions named—were cases in which pyrexia at the time of making the observations was absent. They were for the most part cases of subacute or chronic disease, in which the temperature had subsided under sanatorial or climatic treatment.

Our method consisted in making *daily* examinations of the blood of each patient for a short term previous to the commencement of inoculations, and then daily after the inoculations of tuberculin had been begun. This method possesses certain advantages over the less laborious method of estimating the Opsonic Index at the longer intervals usually followed by others. These advantages will become apparent as we proceed. The total number of blood estimations made during the period covered by this paper is 1750. As four hourly temperature records with clinical notes added from time to time were kept throughout the period during which the patients were under observation, it has been possible in some instances to form an opinion as to the relationship existing between inoculations of tuberculin, variations in the Opsonic Index of the blood, and the more common clinical symptoms and signs usually observed in course of the disease.

In considering how best to present the facts observed, the method of "question and answer" seemed to the writers to be superior to other forms, and therefore that mode has been adopted.

Question 1.

We first proceed to consider the subject opened up by question number one, namely, "Does any difference exist between the Opsonic Indices of the blood in (*a*) healthy persons, (*b*) persons who are suffering from pulmonary tuberculosis (not in an acute form), and (*c*) persons who have suffered from pulmonary tuberculosis and are now cured.

In Table I., through the kindness of Dr Bulloch, we are able to present you with the result of the examinations of the blood of a large number of healthy persons in addition to those obtained by us. Considering our own figures first, your attention is directed to the fact that whilst a marked degree of variation in the level of the Opsonic Indices recorded obtains, it is a variation within clearly defined limits, namely, between 0.9 and 1.2. The result of Bulloch's observations on the same lines practically coincides with our own, only two out of eighty-six cases registering so low as .8 ; and, as he points out, one of them at least was the case of an anæmic nurse whose condition of health was fairly open to question. So that we may take it, that for all practical purposes the Opsonic Index of a healthy person in relationship to the tubercle bacillus does not vary beyond the limits of 0.9 and 1.2.

Our next inquiry was made in connection with the blood of patients who at one time were known to be the subjects of tuberculous lung disease, and who had apparently recovered. For this purpose we selected thirty persons who had passed through our hands as patients, and who, having fulfilled all the known tests of being cured, are now considered useful lives. Here we were at once struck by the fact that although to all outward appearance these persons were as healthy as those dealt with under the previous heading, their indices differed materially from those of the others. In Table II. (column 2) you will find that not one-half come within the limits of normal health, and that so high a proportion as one-third of the total number are to be found below the .9 — the lower health limit. Notwithstanding their apparent good health, the blood of those subjects is singularly deficient in protective substances in relation to the tubercle bacillus.

Continuing the line of inquiry, we next examined the blood of twenty-five persons undoubtedly suffering from pul-

I.

The Opsonic Indices of the Bloods of 25 *Healthy Persons.*

E. B.	...	1.4		
D. L.	...	1.2		
L. S.	...	1.2		
T. C. L.	...	1.2		
W. S.	...	1.2		
J. C.	...	1.2		
J. G.	...	1.1		
N. J.	...	1.1		
C. C.	...	1.1		
R. L.	...	1.1		
H. M.	...	1.1		
W. T.	...	1	...	.9 to 1.2 (country)
N. S.	...	1	...	
N. H.	...	1	...	Average . 1.0
J. M.	...	1		
W. T.	...	1		
A. T.	...	1		
W. C.	...	1		
N. M.	...	.9		
G. C.	...	.9		
W. O.	...	.9		
W. O. J.	...	.9		
R. J.	...	.9		
J. G.	...	.9		
J. R.	...	.9		

Dr Bulloch's 84 cases . . . . .8 to 1.2 (town)

Average .96

Limits of sound health . . .9 to 1.2

II.

The Opsonic Indices of the Blood of Thirty Cases of Pulmonary Tuberculosis cured in Sanatoria without Inoculation.

Case.	Interval elapsed since patient discharged from Sanatorium. Years.	Months.	Opsonic Index.
1	3	6	·5
2	1	9	·5
3	3	2	.6
4	—	9	·7
5	2	2	·7
6	3	1	·7
7	—	8	·7
8	—	7	·7
9	2	6	.8
10	—	6	.8
11	4	3	·9
12	—	4	·9
13	4	4	·9
14	1	8	1.0
15	3	6	1.0
16	3	6	1.0
17	1	1	1.0
18	1	9	1.1
19	1	8	1.1
20	1	6	1.1
21	—	9	1.1
22	—	6	1.2
23	3	7	1.2
24	4	2	1.2
25	1	10	1·6
26	4	3	1.7
27	3	6	1.7
28	—	6	1.8
29	1	2	2.
30	1	2	2.1

III.

Opsonic Indices of the Blood in Twenty-five Cases of Pulmonary Tuberculosis (Subacute or Chronic).

Case.	Sex.	Age.	Lobes.	Opsonic Index.
1	M.	25	2	1.0
2	F.	22	2	.9
3	M.	28	3	.9
4	M.	28	4	.8
5	M.	52	4	.8
6	M.	27	3	.8
7	F.	26	2	.8
8	M.	28	1	.8
9	F.	26	4	.8
10	M.	—	3	.8
11	F.	26	3	.7
12	M.	29	3	.7
13	M.	52	3	.7
14	F.	18	2	.7
*15	F.	34	Miliary	.7
16	M.	28	4	.7
17	M.	24	2	.7
18	M.	28	2	.7
19	M.	27	2	.7
20	M.	36	4	.7
21	M.	36	5	.6
22	F.	32	1	.6
23	F.	24	3	.6
24	M.	48	2	.5
*25	M.	27	Miliary	.5
		Average		.72

* Terminal miliary.

monary tuberculosis, and at the time of examination undergoing sanatorium treatment. Here (Table III.) in three instances an index was present sufficiently high to bring it within the limits of normal health, but in the remainder of the cases the level of the index was lower than that associated with health.

We thus arrived at the conclusion that whilst there is little difference between the Opsonic Indices of persons under classes two and three, the difference between these and class one—the healthy persons—is definite and characteristic. They are invariably lower than those in health.

As Wright has elsewhere suggested, this difference in the Opsonic Index may be turned to practical account in cases where there is doubt as to the diagnosis, an Opsonic Index beyond the limits of health, that is either above 1.2 or below 0.9, being regarded as highly suggestive of the presence of tubercular invasion.

A case of some interest was one in which there existed some doubt as to the diagnosis. There was a history of slight continued pyrexia which subsided two weeks after admission to the sanatorium. Only very occasional slight cough was present. No sputum was available for bacteriological examination, and the physical signs at the right apex were suggestive of early infiltration. Our suspicions as to the presence of lesion were strengthened by the appearance seen on X-ray examination. It occurred to us that an examination of the blood might prove of assistance. This was made, and the abnormally high index of 1.5 was found to be present. The suspicion thus further increased was shown to be well founded, when about a week afterwards expectoration appeared and bacilli were found in considerable numbers.

Question 2.

The next question which we set ourselves to answer is this, Does any difference exist in the behaviour of the Opsonic Indices of blood in healthy persons and in persons suffering from tuberculosis respectively after inoculation of small doses of tuberculin? In order to elucidate this point we wished to inoculate tuberculin (T.R.) in small amounts into healthy persons, and into persons suffering from pulmonary tuberculosis respectively; but, for obvious reasons, a difficulty presented itself in the shape of securing an adequate supply

of healthy subjects. In addition to the results of inoculating healthy subjects obtained in ourselves, we have succeeded in obtaining the results secured in two other cases of healthy members of the profession. In so far as the inoculations were not followed by any sign of lowering of the protective substances in our sera, we had no room for anxiety. Indeed, exactly the opposite phenomena ensued. Our indices in each case immediately rose, and from being 1.0 and 1.1 previous to inoculation, they speedily attained high levels—in one case reaching to 2.1. This feature, then, is common to the observations made on healthy subjects, so far as we have been able to secure any: after tuberculin inoculation the level of the Opsonic Index does not fall, but on the contrary immediately rises.

In marked contrast to this stands the behaviour of the opsonic Indices of twenty-five patients suffering from pulmonary tuberculosis, ascertained immediately after inoculation with small doses of tuberculin (T.R.). The cases (Table IV.), with the exception of those marked "miliary," were at the time of inoculation all apyrexial. You will observe that in each instance—there is no exception to the rule—a distinct fall succeeded the inoculation. The period covered by this fall Wright first designated by the term "negative phase."

There seems, then, this distinction between the behaviour of the blood of healthy persons and that of persons suffering from pulmonary tuberculosis, that, whilst in the case of the latter inoculation of small amounts of tuberculin (T.R.) is at once followed by the establishment of a negative phase, no such phenomenon appears in the former.

There seems to be a practical application of this observation in relation to diagnosis. The appearance of a negative phase after inoculation of a small quantity of tuberculin (T.R.) may enable one to distinguish between the blood of a subject of tubercular invasion and that of a person in sound health.

Question 3.

Assuming the correctness of the conclusions already arrived at, and the value of the indications given by the presence of the negative phase after inoculation of tuberculin, it seemed to us extremely desirable that a shorter and less laborious method of recognising the incidence and duration of

Table showing Twenty-five Consecutive Cases of Pulmonary Tuberculosis, in which Negative Phase followed the Inoculations of Small Doses of Tuberculin (T.R.).

DAY	A.	B.	C.	D.	E.	F.	G.	H.	I.	J.	K.	L.	M.	N.	O.	P.	Q.	R.	S.	T.	U.	V.	W.	X.	Y.
1	**.9**	**.7**	**.4**	**.8**	**7.**	**.8**	*1.2	**.9**	**.5**	**.8**	**1.**	*1.	**.6**	*1.1	**1.**	**2.1**	**1.3**	**1.3**	*1.2	**.6**	**.5**	*1.1	**.1**	**1.3**	.7
2	.8	.4	.2	.4	.4	.7	.9	.8	.9	.6	—	—	.4	—	.4	—	.5	.5	**1.2**	.5	.3	.8	1.	.9	.6
3	.4	.8	.3	.8	.3	—	1.3	.8	.5	.6	.4	.6	.4	.6	1.	1.1	1.3	1.3	—	.4	.8	1.1	1.	1.2	.8
4	.5	.9	.9	.8	—	1.2	.4	—	1.	—	.4	.9	1.2	.9	.8	.7	1.4	1.3	1.1	.9	1.1	1.4	1.	.7	.6
5	.8	.8	.6	.9	—	.6	1.2	1.	.5	.5	1.	.9	1.2	1.	—	1.6	.7	—	1.	.9	—	1.	.8	1.2	1.
6	.8	.8	**.7**	.7	1.	.6	**1.2**	.6	.8	.6	1.	.7	—	.7	.9	1.6	1.5	1.	1.4	.9	1.	.7	1.4	1.2	.4
7	.8	.8	.4	1.1	1.7	1.4	1.2	—	—	.6	1.3	1.	.7	.7	1.5	1.3	1.2	.8	1.3	—	1.	1.	1.5	1.3	1.2
8	1.	1.	.6	.8	**.9**	1.	1.2	1.2	1.	1.	—	.9	**.7**	1.3	1.5	.7	—	1.5	1.3	.7	.5	1.	1.5	.8	**.9**
9	1.3	1.	.6	**.9**	.8	2.3	1.8	**1.1**	1.	.8	—	.8	.6	**1.3**	1.1	1.1	1.6	.8	1.6	1.2	1.2	1.3	1.5	**1.4**	.6
10	**1.**	.9	.5	.8	1.1	—	**1.3**	—	1.	**1.**	—	.7	.7	1.	1.5	1.4	1.2	1.	—	.5	—	1.4	1.5	.9	.9
11	.3	**.8**	.6	.8	.8	1.2	1.3	.9	1.1	.7	—	.8	1.2	.9	.9	1.8	.8	.9	1.4	—	—	1.5	1.8	1.2	1.4
12	1.3	—	.7	.7	.8	1.6	.5	.9	1.3	—	—	.8	—	1.	—	1.2	.9	—	1.3	—	—	**1.2**	1.4	1.2	.7
13	1.2	—	1.1	.3	.8	—	1.3	.8	1.1	.4	—	.5	—	1.7	**1.4**	—	1.2	1.	**17**	—	—	1.4	2.2	1.2	.9
14	1.2	—	**1.**	1.	.9	1.2	1.4	.6	1.2	.4	—	1.5	—	—	.6	1.4	1.	2.1	—	—	—	1.	**1.4**	1.1	.

* Negative absent on first but present on second inoculation.
(The darker figures signify that the patient was inoculated at this point.)

negative phase should be discovered than by routine blood examinations. In this connection we may say at once that observation of the behaviour of the respiration and pulse yielded us no light whatever. Others made upon the relationship existing between the negative phase and variations in the temperature, however, proved more profitable.

With the object of ascertaining the relative incidence of negative phase and temperature disturbance after inoculation had taken place, with a view to assisting us in arriving at a diagnosis, we placed twenty-five cases of well-marked pulmonary tuberculosis under four-hourly temperature records and inoculated them with a small dose of tuberculin.

The result of this inquiry is seen (Table V.), where it is shown that whilst a negative phase was obtained on first inoculation of sixteen cases, only in four instances did the temperature show any disturbance. In second inoculations necessitated by failure to obtain negative phase on first inoculating, the comparison still favours the negative phase. If we use, as was done here, three inoculations, we discover that while temperature disturbance falls to be noted in only 47 per cent. of the cases, the negative phase is present in 100 per cent. When Koch's old tuberculin was introduced, we were told to base our conclusions as to the presence or absence of tuberculosis upon the temperature reaction which followed inoculation of that tuberculin. It is here shown there are many instances in the cases dealt with, amounting to over 50 per cent., where, although tuberculosis is undoubtedly present, the temperature reaction, after inoculation with the T.R. preparation, gives no indication of its presence whatever, negative phase being present in every instance. Temperature disturbance, therefore, is not to be relied upon. The establishment of a negative phase, on the other hand, as compared with temperature disturbance, is a safe and reliable guide in diagnosis of pulmonary tuberculosis.

In course of our work we observed eleven instances in which establishment of negative phase coincided with rise in temperature ; we leave out of consideration those few instances in which it appeared to be accompanied by a fall in the temperature curve. We examined these minutely with a view to discovering whether it would have been possible, by carefully watching this pyrexial manifestation, to have rightly

V.

Table showing the Comparative Incidence of Negative Phase and Temperature Disturbance in Twenty-five Cases of Pulmonary Tuberculosis after Three Injections of Tuberculin (T.R.).

Negative phase.			Temperature.		
18	6	1	4	5	2
100 per cent.			47 per cent.		

Thus, whilst temperature disturbance—the guide recommended to us by Koch in using his old tuberculin—is in this instance absent in over 50 per cent. of cases of undoubted tuberculosis, the negative phase is present in every instance, namely, 100 per cent.

VI.

Table showing the Incidence and Duration of the Negative Phase produced by Inoculation of Small Doses of Tuberculin (T.R.) during Treatment of Pulmonary Tuberculosis (120 instances).

No negative phase.	Persistent negative phase.	Day	1	2	3	4	5	6	7	8	9	10	11	12	13	14
			14	12	10	5	7	4	10	4	4	3	4	1	1	5
15	21		84													

gauged the duration of the negative phase. In two instances the duration of the negative phase and the pyrexial disturbance coincided, otherwise the duration of the temperature exceeded and was exceeded by that of the negative phase equally often. The temperature reaction when present was slight, and in the majority of instances, did not exceed three days. We were, therefore, reluctantly forced to conclude that, alike as an indication of the incidence and of the duration of the negative phase, temperature disturbance is of no value whatever.

Question IV.

We come now to deal with the last question, and what is after all the most important question which we have set ourselves to answer in this paper, viz., To what practical use can these facts be put in the treatment of pulmonary tuberculosis?

If the observation of the negative phase is important in relation to diagnosis, much more important is its relationship to treatment. Assuming the accuracy of the proposition, for which there are good grounds, that inoculation of tuberculin reinforces that phase which is present at the time of inoculation, and that, therefore, to inoculate during the negative phase is to increase the manufacture of toxins, and to inoculate during the positive phase is to produce in increasing amounts protective substances, then it becomes a matter of paramount importance to ascertain exactly the state of the blood before we inject tuberculin. We have taken 105 cases of inoculation of patients under treatment in which daily observations of the state of the blood were made before and after inoculation had taken place. The result of an analysis of these is shown in Table VI. In fifteen instances no negative phase occurred. In twenty-two cases a persistent negative phase—that is, a negative phase extending beyond fourteen days—was shown, and the duration of the negative phase in the remaining eighty-four cases varied, as shown in the diagram. Taking these figures as they stand, it may be profitable to consider what would have occurred had inoculation taken place by routine—say, twice weekly. If all the patients had been reinoculated on the third day, in forty-six instances, in so far as they would in that case have been reinoculated whilst the

VII.

Showing the Proportion of Instances in which Inoculation made on various days would in the foregoing 84 *Cases have taken place during the Negative Phase.*

Inoculation on the		Negative phase.		Percentage.
3rd day	...	46	...	55
7th day	...	20	...	24
10th day	...	9	...	11
13th day	...	5	...	6
After 14th day	...	4	...	5

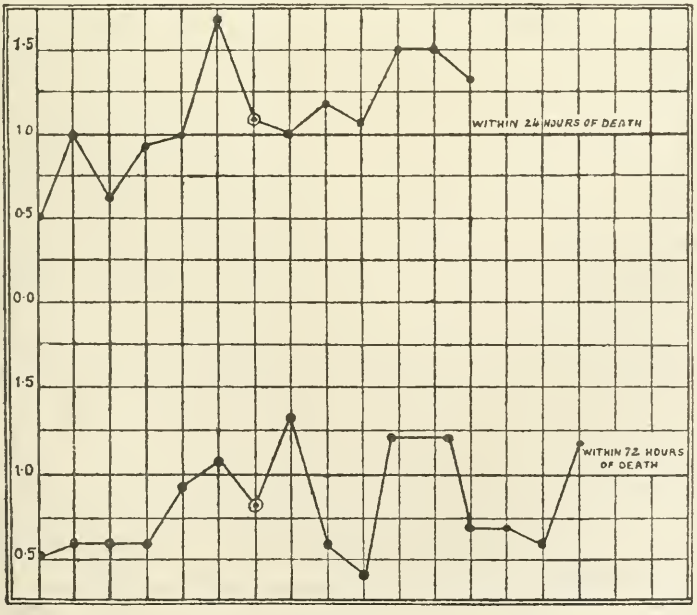
Thus, reinoculation on the 8th day would have avoided the negative phase in 77 per cent. of instances, or in rather more than three out of four instances (modify).

Daily observation of the Opsonic Index of the blood, by enabling one to avoid inoculating during the negative phase, would have prevented harm being done in the remaining one-fourth of the cases.

* Fractions ignored.

VIII.

Opsonic Indices taken Daily during the Terminal Miliary Stage of Pulmonary Tuberculosis.



negative phase was still present, the inoculation, instead of doing good, would probably have done harm. Similarly, on the seventh day, twenty would probably have derived harm, and so on, as in Table VIII., in direct proportion as we increase the period between the injections, until at the end of the fortnight the risk is reduced from 55 per cent. at three days to 4 per cent.

We have therefore in daily observation of the Opsonic Index, an invaluable guide which enables us to do what can be done by no other known means, namely, avoid repeating the inoculation at that time, when, instead of exerting a beneficent action upon the course of the disease it will promote the furtherance of toxic processes.

The next point which we wish to make clear is this, Can clinicians by this method of treatment benefit a patient after sanatorium and climatic treatment has entirely or partially failed? As examples of cases where sanatorium treatment failed, it is convenient to consider two cases of miliary tuberculosis. Needless to say, both did badly, indeed, both died. We may say that tuberculin inoculations were given at the urgent request of friends, and with the full approval of the consultants, who saw both patients at a late stage of the disease. Although the record of the Opsonic Index (IX.) brings us up to very nearly the time of death, in one case to within thirty-six hours, and in the other to within seventy-two hours of death, in neither case is it very low, indeed, standing much higher than those of some cured patients, each is surprisingly high. Reduced to chart form (Table VIII.) both share this in common, that they show large variations, at times reaching above normal and at times passing below. Their opsonic charts resemble their temperature charts in the irregularity and the amplitude of the curve which they present. Simply, therefore, for the opsonic index to reach a high level from time to time does not necessarily imply that improvement is taking place. In these cases inoculation appeared to do no good. Your attention is next directed to its action in another type of case.

As has been already pointed out, the majority of cases dealt with are of long standing, and as they are for the most part under treatment now, we are not in a position to state the ultimate result obtained in those cases. Some of those patients had spent years abroad at health resorts of the

highest repute, and had supplemented that course by undergoing terms of sanatorium treatment in this country. It was at the end of such treatment that the bloods were examined, and here (Table IX.) you observe that uniformly they showed low Opsonic Indices—indices well below normal in over twenty out of twenty-five cases. These patients elected to have inoculations of T.R. tuberculin administered by this method, and on Chart X. there is recorded the state of their Opsonic Indices after having undergone a period of not more than six weeks of such treatment. The chart also shows in each case the amount of tuberculin which they had received. These figures, we may say, were arrived at by taking an average of three consecutive readings, care being taken in making the estimation to avoid the negative phase. All other elements in the treatment having remained the same, the rise which had taken place in the supply of protective substances present in the serum which these higher indices point to can only be accounted for by the action of the tuberculin. It is, therefore, plain that in inoculations given in this way we have a valuable agent, by means of which the resistance to, and, therefore, the power of combating the power of the disease, can be raised to a higher level than has been in these cases obtainable by other means. Diagram XI. furnishes a survey of a case successfully treated in this way in which recovery was eventually secured after the condition had become, to all appearances, stationary after prolonged climatic and sanatorium treatment.

Consideration of the Opsonic Curves in cases which have run a favourable course reveals two points of interest. First they show how inoculation by this method may be employed in order to gradually raise the resistance of a patient suffering from tuberculosis. The period corresponding to the negative phase is jealously avoided, and a point slightly higher than the last one at which inoculation took place is selected for successive inoculations until a level is reached well above normal. Secondly, the condition compatible with recovery is found to be not an index standing at a very high level, but one slightly above normal and maintained with slight variation at that level. Indeed, the analogy between the opsonic curves and the temperature curves present respectively in active and in arrested disease is strikingly similar. The temperature curve

IX.

The Opsonic Indices of 25 Cases of Pulmonary Tuberculosis after varying Terms of Treatment (without Tuberculin Inoculations).

Name.	Duration of disease.		Duration of treatment.				Opsonic Index.
			Climatic.		Sanatorium.		
	Years.	Months.	Years.	Months.	Years.	Months.	
M.	2	6	1	—	1	—	1.1
K.	1	2	—	—	1	2	1.
L.	—	5	—	—	—	5	.9
H.	—	5	—	—	—	2	.6
M'C.	4	3	—	—	1	7	.9
M.	1	—	—	—	—	2	.8
L.	4	6	4	—	—	—	.8
M'D.	3	10	2	4	1	4	.8
F.	3	—	—	—	2	3	.8
W.	1	5	—	2	—	10	.8
G.	4	—	—	—	—	2	.8
T.	—	2	—	—	—	2	.8
P.	4	3	—	—	—	1	.7
M'A.	4	1	2	6	1	1	.7
M.	7	—	1	—	1	2	.7
H.	8	—	6	—	—	6	.7
A.	1	2	—	—	—	9	.7
B.	1	9	—	—	—	10	.7
B.	2	—	—	3	1	4	.7
M.	1	—	—	—	—	4	.7
K.	—	4	—	—	—	4	.7
W.	3	—	—	—	1	4	.5
A.	3	6	—	—	2	2	.5
S.	1	5	—	1	1	—	.5
G.	2	4	—	—	1	10	.5

X.

Comparison of the Opsonic Indices of the Bloods of Twenty-three Patients, taken after a term of Sanatorium Treatment only, with those taken after a short term of Sanatorium plus Tuberculin (T. R.) Inoculations Treatment.

Name.	O. I. before inoculation.	Doses given within a period of 5 weeks.	O. I. after inoculation.
S.	·5	4 of $\frac{1}{500}$	1.5
P.	·7	4 of $\frac{1}{500}$	1.3
M'A.	·7	4 of $\frac{1}{500}$	1.1
M'D.	.8	4 of $\frac{1}{500}$	1.
W.	·5	5 of $\frac{1}{500}$	1.1
A.*	·5	1 of $\frac{1}{500}$, 1 of $\frac{1}{1000}$	.8
M.	·7	3 of $\frac{1}{500}$	1.3
B.	·7	3 of $\frac{1}{1000}$	1.2
F.	.8	3 of $\frac{1}{500}$, 1 of $\frac{1}{50}$, 1 of $\frac{1}{500}$	1.3
A.	·7	3 of $\frac{1}{500}$	1.2
L.	·9	3 of $\frac{1}{1000}$	1.2
W.	.8	1 of $\frac{1}{1000}$	1.1
K.	1.	1 of $\frac{1}{1000}$	1.1
H.	·9	3 of $\frac{1}{1000}$	1.4
B.	·7	4 of $\frac{1}{500}$	1.3
M.	·7	1 of $\frac{1}{1000}$, 1 of $\frac{1}{2000}$, 1 of $\frac{1}{1000}$	1.5
G.	·5	4 of $\frac{1}{500}$, 1 of $\frac{1}{400}$	1.1
H.	·7	2 of $\frac{1}{1000}$	1.
G.	.8	3 of $\frac{1}{1000}$	1.
K.	·7	4 of $\frac{1}{1000}$	1.3
M'C.	·9	3 of $\frac{1}{1000}$	1.4
T.	.8	2 of $\frac{1}{500}$	1.2
M.	.8	3 of $\frac{1}{50}$	1.4

* Spinal complication. Index eventually rose above 1.0.

of active disease is one which oscillates as a rule through a large range, similarly with the opsonic curve. And just as we find in arrested disease a temperature curve of small range existing in the vicinity of normal, so we find in such cases an opsonic curve, usually above 1.0, and varying not more than two or three points from time to time.

Although the last chart which it is proposed to submit for your consideration is not a record of work done by ourselves, the interest which it arouses is so closely connected with the subject under consideration that we do not feel any apology is necessary in quoting it. As will be observed by referring to Table No. XII., Trudeau is able to furnish us with comparative results obtained in a body of work, extending over twelve years, of cases treated respectively by sanatoria treatment alone and by sanatoria treatment plus inoculations of tuberculin. At the time of writing, no less than 20 per cent. more of the cases which had been treated by tuberculin and sanatoria measures were alive and well, than of those cases in which no sanatorial measures alone had been followed.

In order to meet the obvious objection raised by hostile critics to the validity of his conclusions that "no doubt the cases treated by tuberculin had been carefully selected because of the favourable features they presented, and that no such selection had been made of cases treated by sanatoria measures alone," he supplied us with the results obtained in another series of cases detailed under letter B of the same chart.

Here it is shown that of cases in all respects, so far as could be judged, of the same type and at the same stage of the disease, an advantage of over 15 per cent. is present on the side of those treated by tuberculin. Assuming the correctness of the observations made by Trudeau, it seems that in those cases in which tuberculin was employed, not only was the percentage of the cures increased, but the liability to relapse was diminished. Is the explanation not to be found in the fact that, even handicapped as he was by the absence of any precise and scientific method to guide him in his work, such as is to be found in the systematic observations of the Opsonin Index, he succeeded, by means of tuberculin inoculations, in raising the resistance of the cases so treated to a higher level than was reached in those cases in which he depended entirely upon sanatorial measures of treatment? If that is so, then

XI.

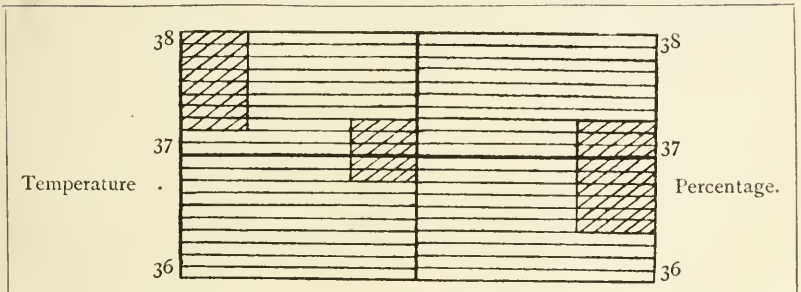
Male, æt. 26. Three-lobed lesions (bilateral).

TREATMENT.

Sanatorium + 11 months. Climatic 3 years.

Sanatorium + inoculation 4 months.

March, 1904. February 10th, 1905. June 10th, 1905.



Weight . . .	61.750 kilos.	65,800.	68.800.	4.5 per cent.
Hb.	78.		90. 100.	11.1 per cent.
R. B. C. . . .	4,700,000.	4,100,000.	4,960,000.	20.9 per cent.
Opsonic Index .		.86.	1.46.	6.9 per cent.
Sputum, amount	Trace.	Trace.	0.	—
T. B.	Present in fair nos.		0.	—
Exercise . . .	0.	6 miles.	10 miles.	—
Physical signs .			Occasional post-tussive crep. remains in supra-clav. area.	—

XII.

Trudeau's Results from 1890-1901.

A.

Cases treated (non-tuberculin).		Cases treated (tuberculin).		Advantage to tuberculin cases.
1367	...	143		
Alive. 38 per cent.	...	Alive. 58 per cent.	...	20 per cent.
Dead. 39.6 per cent.	...	Dead. 33 per cent.	...	Balance untraced.

B.

Incipient cases only.

Alive. 61 per cent.	...	Alive. 76.7 per cent.	...	15.7 per cent.

there seems every likelihood that clinicians, by means of this line of treatment, will in the near future be able to obtain results in the treatment of tubercular disease which will be superior to any which had been obtained in the past. In concluding the report on these investigations, we wish to place on record our sense of indebtedness to Dr Wright for the privilege enjoyed in working for a time in St Mary's Pathological Laboratory under his personal supervision, and for much valuable guidance and help received at his hand from time to time in connection with our work.

Meeting II.—December 6th, 1905

Dr J. O. Affleck, M.D., *President, in the Chair*

I. Election of Members

Lewis Beesley, F.R.C.S. Ed., L.R.C.P. Ed., St Leonards-on-Sea; A. Fleming, M.B., Ch.B., Corstorphine; J. S. Fraser, M.B., F.R.C.S. Ed., 13 Heriot Row; A. M. Malcolmson, M.B., Ch.B., Corstorphine; Major D. G. Marshall, M.B., 25 Chester Street; J. H. Harvey Pirie, M.B., Ch.B., 13 Alva Street; D. G. Reid, M.B., Ch.B., 16 Greenbank Terrace; A. J. M. Spiganovicz, M.B., Ch.B., 1 Seton Place; James William Somerville, M.D., D.P.H., Galashiels; and F. D. Simpson, M.D., F.R.C.S. Ed., 7 Kew Terrace, were elected Ordinary Members of the Society.

II. Exhibition of Patients

1. *Mr C. W. Cathcart* exhibited—

(*a*) A girl aged four, who had suffered from CONGENITAL DISLOCATION OF THE LEFT HIP. A year ago the dislocation was reduced by Lorenz's method, and the limb had been fixed in plaster until a fortnight ago. The plaster case had been renewed several times, and at each change the limb had been brought more nearly into the normal position. Following a suggestion kindly given to him by Mr Stiles, Mr Cathcart had at first fixed the sound as well as the affected limb in the

abducted position. The affected limb was still slightly abducted and everted, and the movements of the hip-joint were restricted, but the child was running about and was improving daily in freedom of movement. An X-ray negative, taken recently, showed that the head of the femur was in the acetabulum. On palpation from the front some thickening was felt in the neighbourhood of the acetabulum, where, before reduction, there had been an absence of resistance as compared with the other side.

(*b*) A MAN WHOSE LEFT PATELLA HAD BEEN TWICE SUTURED FOR FRACTURE.

James C., aged twenty-six, was admitted to Ward 18, Royal Infirmary, on January 20th, 1905, suffering from a fracture of the left patella, produced by muscular action. The knee was much distended by blood clot, and the ends of the bone separated by a considerable interval. Under chloroform the front of the knee-joint was exposed by a curved incision, with its long axis vertical. Large masses of firm clot were removed from the interior of the joint and from the interval between the broken surfaces. Embedded in the clot in the latter portion were thick strands of fibrous tissue, torn from the surface of the lower portion of the patella. The broken surfaces were placed in apposition, and held there by five chromicised catgut sutures passed through the adjacent soft structures.

The patient made a good recovery. Although some stiffness of the knee remained, he returned to work.

On August 3, 1905, while at work, he was helping to lift a heavy weight, and the fractured patella suddenly snapped again.

He was again admitted to Ward 18, and the knee-joint was reopened through the scar of the first operation. On this occasion the ends of the bone were united by two silver wire sutures, passed vertically, but not reaching to the articular surface. Passive movement was begun within a few days of the operation. The wound healed by first intention, and the bone united rapidly.

The movements of the joint are now as good as on the other side, so that the second fracture and suture has been certainly no disadvantage.

The stiffness after the first operation was probably due to delay in moving the joint from fear of the catgut stitches

giving way. The wire gave a greater sense of security in this respect, and encouraged earlier passive movement.

2. *Dr Byrom Bramwell* exhibited—

(*a*) A CASE OF MULTIPLE LIPOMATA WHICH WAS HEREDITARY. The patient, a man aged fifty-one, a miner by occupation, was admitted to the Edinburgh Royal Infirmary on account of symptoms suggestive of an abdominal aneurism. On examination it was found that he was the subject of multiple lipomata. Innumerable fatty tumours were situated in the subcutaneous tissues. They varied in size from a small cherry to a large hen's egg; they were most numerous on the forearms, thighs, and buttocks, but were also present in considerable numbers on the upper arms, legs, and trunk; there were none on the face, scalp, hands, or feet. The tumours were pure lipomata. This had been proved by microscopical examination: Dr Shennan had examined one of the tumours which Mr Caird had kindly removed. The patient stated that the condition had been present as long as he could remember—probably since birth. The tumours caused him no inconvenience and were not painful; this was an important point, for in some cases of multiple lipomata there is great pain. The term "adiposis dolorosa" has been given to that condition—multiple symmetrical fatty tumours in the subcutaneous tissues associated with severe neuralgic pains. It was first described by Dr Dercum of Philadelphia, and is consequently sometimes termed Dercum's disease. Cases of adiposis dolorosa are chiefly (? exclusively) met with in women, usually middle-aged women. The exact pathology of the condition is not known, though in several cases atrophic changes in the thyroid gland have been found. Dr Dercum has seen marked improvement result from the administration of thyroid extract in cases of this description. Adiposis dolorosa is a rare disease, but one or two cases had come under Dr Bramwell's notice. The present case was an example of simple multiple lipomata; it is a very rare condition, for in most of the cases in which multiple tumours, which at first sight appear to be lipomata, are found in the subcutaneous tissues, microscopic examination shows that the tumours are fibromata or neuromata. Another point of great interest in this case was that the disease, if it should be termed a disease,

is hereditary : the patient's mother had the same condition ; in her case, as in the patient's case, the subcutaneous tumours were chiefly situated in the forearms and thighs. Dr Bramwell had not been able to find any other case of multiple lipomata in which the condition was hereditary. So far as he had been able to ascertain, none of the other members of the patient's family (father, two brothers, six sisters, and their numerous children) had been affected. The patient himself has six children ; they are all healthy. Dr Bramwell had not been able to get exact details regarding his mother's relatives, but so far as is known none of them were affected.

(*b*) A case of MARKED ATAXIA OF THE LOWER EXTREMITIES, without any loss of the sense of movement (muscular sense).

The patient, a fireman, aged 60, was admitted to the Edinburgh Royal Infirmary on October 28th, suffering from weakness, pain in the back, and difficulty in walking. His gait was very ataxic ; he threw out his legs in an incoordinate way, bringing his heels down on the ground with a thud ; he had great difficulty in turning and in getting up and sitting down ; in walking he raised his legs higher than most patients with tabes do ; there seemed to be an element of "steppage" in the case. On examination, nothing was detected except the ataxia ; the knee-jerks and Achillis-jerks were active ; the Argyll-Robertson condition of the pupils was not present. There were no lightning pains ; no affection of the bladder or rectum ; no thoracic, ulnar, or other analgesia ; there was no history of syphilis. The case was obviously not one of tabes.

The interesting point in the case was that with such extreme ataxia there was no derangement of the sense of movement (muscular sense) ; Rombergism was slight ; the patient could correctly tell the direction of passive movements of the toes, ankle, knee, hip, when the eyes were closed ; he could correctly estimate the difference of slight weights attached to the lower extremities when the eyes were closed ; he could correctly tell the position of the limbs after passive movements when the eyes were closed. There was no paralysis. The exact nature of the case was not clear. The patient's age seemed to be against a mere functional condition. Dr Bramwell had never seen a case in which there was such extreme ataxia without some derangement of the sense of movement (muscular sense).

3. *Mr E. S. Carmichael* exhibited a child, æt. 18 months, which had been operated on four months previously for TUBERCULOUS STRICTURE OF THE ILEUM. The patient had had symptoms of chronic obstruction for some months previous to admission.

There was marked distension of abdomen, well-marked ladder pattern, constipation and attacks of vomiting, and increasing general emaciation.

An incision was made over the cæcal region, and two strictures found within four inches of the ileo-cæcal junction. The bowel above the stricture was markedly hypertrophied. The ileo-cæcal glands were tuberculous, and the cæcum and ascending colon atrophic. An artificial anus was made immediately above the seat of stricture.

Ten days afterwards the artificial anus, along with the cæcum, ascending colon, and ileo-cæcal glands were removed, and an end-to-end anastomosis of the ileum and transverse colon performed.

The child made an uninterrupted recovery, and in spite of an attack of measles had gained 4 lbs. in weight since leaving hospital.

4. *Mr David Wallace* exhibited—

(*a*) Two patients after PROSTATECTOMY BY THE SUPRAPUBIC ROUTE.

(1) Mr F., aged 68 years, had suffered for four years from pain and much frequency of micturition. Difficulty in micturition had at times been considerable, but catheterisation had not been required. The urine contained much pus and mucus, but hæmaturia had been only very occasionally present. Per rectum the prostate was not large, but was firm and the lateral lobes equal.

Suprapubic cystolomy was performed on the 1st of November 1905. Two stones were recovered from the post-prostatic pouch, and the prostatic gland was enucleated in the usual way, but with some difficulty. The prostatic urethra was torn across, but did not come away with the specimen. In six days the urine was passed per urethram, and in three weeks the patient left the surgical home. Mr Wallace attributed the quick passage of urine by the urethra to the fact that the opening in the bladder was situated at a lower level than the

opening in the abdominal wall. The Sprengel pump was discarded on the fifth day.

(2) I. S., aged 68 years, had suffered from frequency of micturition for some years, at least six. One year ago he began to have difficulty in passing his urine, the frequency was much greater than formerly, and in September 1905 retention occurred and catheterisation was required. Since the first catheterisation he had been unable to pass any water without using the catheter, and there had been considerable hæmaturia. Per rectum the prostate was considerably enlarged. It was firm, and equally enlarged.

Suprapubic cystolomy on 25th September. The prostate was enucleated with some difficulty in one mass. The prostatic urethra was torn across. The patient made an uneventful recovery, but there was longer delay than usual in the urine being passed per urethram. This occurred for the first time on the 26th day after the operation. After that date it came wholly by the urethra, and the patient left hospital perfectly recovered on the fifth week.

(*b*) A patient was to have been shown by Mr Wallace, from whom he had removed an ADENO-CARCINOMA OF THE DESCENDING COLON three weeks previously. End-to-end suture was adopted, and the patient, a woman aged 59 years, made an excellent recovery ; but although she was able to walk and was recovering rapidly, he had thought the risk of bringing her to the meeting was not justifiable. He contented himself therefore with merely showing the specimen, which was a very typical example of malign annular stricture of the large intestine.

5. *Mr F. M. Caird* exhibited—

(*a*) CASE OF RECOVERY AFTER TETANUS. Mr G. L., æt. 26, was admitted to the medical wards of the Royal Infirmary on 8th October, suffering from abdominal pain, and symptoms which appeared to be of peritonitic or influenzal origin. For several years similar but less severe attacks had troubled him, associated with diarrhœa and vomiting. On examination, however, the diagnosis of tetanus was established, and traced to a crush of the left middle finger, which he had sustained when lifting a cabinet on 17th September. The finger was dressed, but thereafter neglected,

and the patient continued working for ten days, when he believed that he received a chill which occasioned pain all over his body, shivering, and malaise. On October 6th he had pain in the chest and abdomen, and on the 7th it became paroxysmal, and he sweated profusely. On admission he had marked risus sardonicus, rigid abdomen, and stiff back. The finger was amputated, and the administration of Tizzonis serum in large doses, chloral and bromide of potassium, began. The patient's symptoms increased; he had opisthotonos, spasms of the legs, retention of urine, and bit his tongue, although spasm of the jaw was not marked. On 14th October his pulse was 124 and his temperature 102.6 and respirations 32. From that date a gradual improvement and recovery followed.

(*b*) CASE OF RECOVERY AFTER PERFORATION OF GASTRIC ULCER. Miss K. K., æt. 35, who had for fourteen years suffered from intermittent gastric pains and dyspepsia, had sudden epigastric pains and symptoms of ruptured gastric ulcer. On opening the abdomen, 13 hours after the sudden attack, a perforation was found on the anterior cardiac surface, $2\frac{1}{2}$ inches from the lesser curvature. It was closed, and the pouch of Douglas was washed out. The peritoneal fluid contained streptococcus pyogenes, and staphylococcus. Within nine days a left pleurisy developed, which was tapped, but ultimately empyæma followed, which required resection of a rib for drainage. The patient is now in good health.

(*c*) Case of RECOVERY AFTER PERFORATION OF DUODENAL ULCER.

Mr R. T. had been in perfect health when at noon, on 24th October, he almost fainted because of sudden severe abdominal pain. He had a small thready pulse, subnormal temperature, and fixed abdomen. Within an hour the liver dulness disappeared; there was some distension, and, receiving a dose of morphine, he was sent by rail to the Royal Infirmary. He vomited twice *en route*. Laparotomy, ten hours after symptoms appeared, disclosed a small punched-out aperture at the anterior aspect of the first part of the duodenum, from which a clear alkaline mucus exuded. Much gas escaped, and turbid purulent fluid was washed from out the pouch of Douglas. The ulcer was sutured and the pelvis drained. A good recovery followed.

It is noteworthy that this patient required an operation on

19th July for *appendicitis*, and that he had also had sub-trochanteric coleotomy for marked *coxa vara* with marked benefit.

III. Exhibition of Specimens

1. *Drs Bruce, M'Donald, and Pirie* exhibited photographs of two cases of DUPLICATION OF THE SPINAL CORD.

The first case showed commencing doubling in the first lumbar segment, which was completed in the lower part of the segment. Each part was at first only a half cord, but when traced down to the fourth lumbar segment each became an almost perfect cord, only the two superadded inner cornua were less complete than the outer ones. There were, however, a few motor cells in the anterior cornua, and the posterior received a few root fibres.

In the second sacral segment the two cords again united, but not quite symmetrically, and to the extreme lower end of the filum terminale there were two central canals, one of the cords being represented by an additional mass of grey matter.

The second cord showed commencing doubling in the first sacral segment, completed in the second sacral. In this instance the two cords remained united. There were two central canals, four perfect posterior cornua, but only two complete anterior cornua, the inner being rudimentary. In the fourth sacral segment the doubling again resolved itself into a normal single cord.

In neither case was there any abnormality of the spinal column, or any symptoms attributable to the condition. Full accounts have been published in the *Review of Neurology and Psychiatry*.

2. *Mr C. W. Cathcart* exhibited specimens from a case of MILIARY TUBERCULOSIS, starting from tuberculous kidney.

The patient was a boy aged 13, inmate of an industrial school. In jumping off a lorry in motion he fell inwards and one wheel passed over his pelvis. He was brought into the Infirmary. There were no signs of fractured pelvis, but he passed blood mingled with his urine, and a rupture of the bladder was suspected. Two days afterwards his temperature and pulse rose suddenly, and abdominal respiration became restrained. The abdominal cavity was opened on the supposi-

tion that escape of urine had caused the symptoms. This was not confirmed, but, as a catheter could not be passed, the bladder was first opened above the pubes and afterwards drained from the perineum.

The boy showed no signs of healing. His temperature and pulse kept high, and he sank and died about fourteen days after the operation.

At the post-mortem examination the left kidney was found to be the seat of numerous foci of tuberculosis, some evidently of long standing. The wall of the bladder had tuberculous deposits upon it. *Miliary tuberculosis* was present in the spleen, right lung, liver, right kidney, and on the surface of the peritoneum.

When questioned, the boy and his father denied the history of any previous urinary trouble, but the manager of the school—only seen after the boy's death—said that the boy had been unable to hold his water for long, and frequently wet his bed.

3. *Mr E. S. Carmichael* exhibited—

(*a*) THE CÆCUM, ASCENDING COLON, AND PART ILEUM, along with the ARTIFICIAL ANUS, which had been resected in the child exhibited at the meeting.

(*b*) A VESICAL CALCULUS, removed from a boy æt. 3 years, by suprapubic cystotomy. Mixed uric acid and phosphatic.

4. *Mr David Wallace* exhibited—

(*a*) TWO PROSTATES, enucleated through suprapubic route.

(*b*) MALIGN TUMOUR, adeno-carcinoma of portion of descending colon, removed by colectomy.

5. *Mr F. M. Caird* exhibited a preparation of INTRACRANIAL ABSCESS which lay towards the upper extremity of the fissure of Rolando. It had given rise to symptoms indicating affection of the mouth and arm centres, and was not discovered on trephining. The patient had been operated upon successfully for ruptured gastric ulcer, and thereafter for empyæma, about four months previously.

6. *Mr Alexis Thomson* exhibited a TUBERCULOUS LEFT

KIDNEY removed from a girl æt. 26, who had suffered for four and a half months from frequent and painful micturition. The urine contained pus and blood, and what appeared to be tubercle bacilli. The right kidney was palpably enlarged, but was not sensitive. Examination with the cystoscope showed the left base of the bladder to be obscured and the right urethral opening to present a crateriform appearance. After injection of indigo-carmine into the buttock the urine of the two kidneys was separated by Luy's instrument. From the right side there escaped abundant indigo-stained urine, with a trace of pus and a few hyaline and epithelial casts; that from the left was small in amount, free from indigo-colour, contained blood cells, abundant pus, and no casts. The lower end of each ureter was felt as an indurated cord in the lateral fornix. The diagnosis made was that of advanced tuberculous disease in left kidney, and compensating enlargement of the right kidney, with commencing tuberculous disease of the lower end of both ureters.

The patient made a good recovery.

IV. DEMONSTRATIONS

1. PERSONAL EXPERIENCES IN THE DETECTION OF HUMAN BLOOD BY MEANS OF THE PRECIPITIN SERUM TEST OF UNLENHUTH.

By W. G. AITCHISON ROBERTSON, M.D., D.Sc., F.R.C.P.Ed.,
Lecturer on Medical Jurisprudence, Surgeons' Hall, Edinburgh.

THE detection of blood stains, either in the fresh or old condition, is by no means difficult, the microscopic, chemical, and spectroscopic characters pertaining to them being sufficiently characteristic.

The routine method of examining a stain for the presence of blood is first, after making a solution in distilled water or in normal saline solution, to test it with tincture of guaiacum and ozonic ether, either in the test-tube or on the microscopic slide. Then the character of the corpuscular elements is determined by the microscope. It may be necessary to tease a small portion of the stained fabric on the slide and examine at once. In the case of blood which has been thoroughly dried the

measurement of the corpuscles is of little value, as, owing to their irregular expansion, one would not dare to affirm conclusively from what animal the blood had come. An oval shape or the presence of a nucleus in each corpuscle would of course allow of a differentiation from mammalian blood.

The test by the formation of hæmin crystals would next be tried, and lastly the spectroscopic appearance of the solution would be investigated, the absorption bands due to oxyhæmoglobin, reduced hæmoglobin, methæmoglobin, hæmatin, etc., allowing of easy recognition.

The above methods, however, only allow of the recognition of blood as such, but not of its differentiation as to origin. The precipitin test, as devised by Professor Unlenhuth, affords the only means by which one can determine from what animal the blood has come. The reagent in this case is a specific serum which has to be manufactured for the recognition of each particular blood. In brief, the method of manufacture is as follows: Suppose we wish to obtain a serum which will give a precipitate with human blood alone. Into the peritoneal cavity of a rabbit from 10 to 20 c.c. of human blood, or the same after defibrination or even human blood serum is injected. This is repeated at intervals of four or five days. Gradually the serum of this rabbit's blood increases in precipitating activity until, at the end of the second or third week, it has reached a maximum. The animal is then killed and its blood allowed to flow into a sterile vessel in which it coagulates. From 30 to 40 c.c. of active serum may be obtained from each rabbit.

The test is performed in the following way: The colouring matter from the stain (supposed to be human blood), having been dissolved in a few drops of water, is diluted with normal saline solution in the proportion of 1 : 100 or even 1 : 1000. Two c.c. of this are placed in a very narrow test-tube, and by means of a fine pipette (easily made from an ordinary vaccine tube) a little of the prepared serum from the rabbit is allowed to form a layer beneath the diluted human blood. At the junction of these fluids a white line of opacity forms within a few seconds if the suspected fluid be human blood, but not if the blood be from any other animal. The opaque line increases in density, and soon after the precipitate falls to the bottom of the tube. This action is usually completed within

ten minutes. With the blood of certain apes this reaction also takes place, but it is neither so rapid nor so distinct as with human blood. This is a fallacy which in our own country would hardly ever occur, and even if it did, the error could be eliminated by injecting human blood into the particular variety of ape, and then employing the serum of its blood as a precipitin test for human blood.

As regards my own experience with this serum, I made numerous experiments with human blood in all degrees of dilution, even up to the proportion of 1 : 1000 and beyond, and all gave a precipitin reaction with the prepared serum. Even in cases where the blood had been kept until putrefaction had

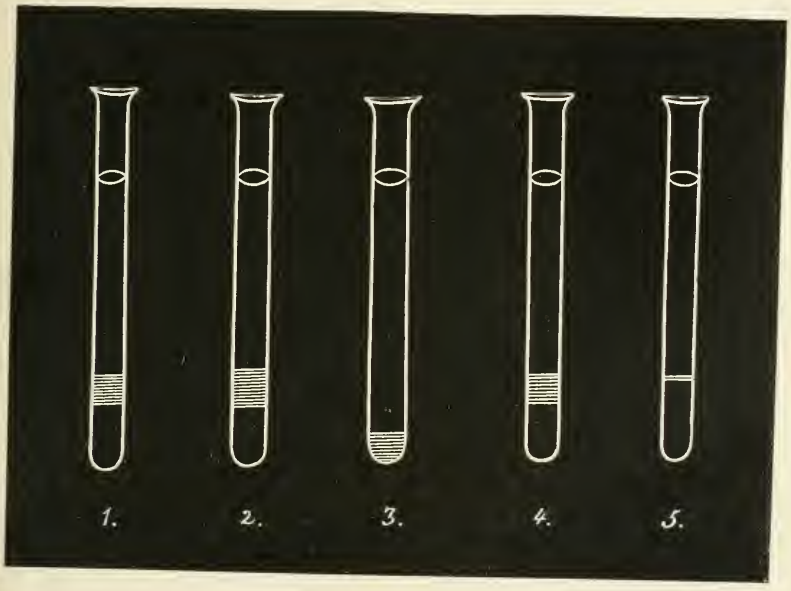


FIG. 1. Reaction with solution from a stain 4 weeks old, after 5 seconds.
,, 2. The same after 12 seconds.
,, 3. The same after 30 minutes.
,, 4. Reaction with stain 9 years old, after 10 minutes.
,, 5. No reaction with blood of ox.

set in, the filtered fluid gave a marked reaction with the specific serum.

In the case of stains on articles of clothing or on instruments, solutions were made with normal saline solution and carefully filtered. I examined numerous stains of very varying ages. Those which were from one to twenty-four weeks old

gave rapid and marked reaction, while in those which had been kept for several years the precipitate formed more slowly, and though quite distinctive was not so marked as in the case of more recent stains. The oldest stain which was examined was nine years old, and the figure in the illustration shows the appearance at the end of ten minutes.

The blood of other animals which might most easily be confounded with human blood, as the ox, sheep, or pig, gave neither in the fresh condition nor as solution from old stains any reaction whatever.

In medico-legal work all that we would be required to determine would be whether the blood was from a human being or not. Unlenhuth's test is the only one which up till the present furnishes us with a positive answer to this question.

If the blood were not of human origin it might be a very difficult matter to tell from what animal it had come, the range is so wide. If the question were narrowed down to whether, for example, it was from a horse, the answer would be easy. We would prepare a rabbit by injecting it with horses' blood, and so obtain a serum having a precipitating action on the blood of the horse alone. In the same way a positive or negative answer could be given in any stated case.

2. *Dr W. T. Ritchie* gave a microscopic demonstration of MICROCOCCUS CATARRHALIS from a case of bronchitis.

V. Original Communications

1. ON THE TREATMENT OF FRACTURED PATELLA BY TRANSVERSE WIRING (WITH A SYNOPSIS OF SIXTEEN CASES)

By George G. Hamilton, M.B., F.R.C.S., Surgeon to the Liverpool Royal Infirmary

(Plates I.—III.)

Four years ago, at a meeting of our Liverpool Medical Society, I spoke *against* the open method of wiring the patella, because at that time, considering the advantages and the risks, I did not think it was *justifiable* as a routine operation. With improved methods, I found I could open the knee-joint for other conditions, without suppuration, and as on this the whole

question of wiring turns, I now believe that the open method quickly performed is the best, at any rate for the working man.

The first surgeon, probably, to wire the patella was Burton of Philadelphia, and, shortly after, Cooper of San Francisco, in 1861. I saw Lord Lister wire his first case in 1877. But even in his hands there was always a grave anxiety, and until recently most surgeons have been content with the results they have obtained by non-operative methods.

It will be profitable to look for one moment at the opinion of leading American, German, French, and English surgeons, expressed as recently as three years ago. Of seventy-one surgeons, chiefly American, replying to an inquiry by Dr Powers, only *nine* practised the open method, and these included Kocher, Lister, Halsted, Horsley, and Phelps. *Forty-one* would operate on selected cases only, and *twenty-one* would not operate.

As representing the French school, Berger, as recently as May 1903, whilst holding that the open suture or encircling ligature remain the best methods, in the absence of decided contra-indications, points out that such treatment, especially when practised by inexperienced surgeons, is not free from complications so serious as to enforce the necessity of not neglecting the older and more simpler methods. Results almost as good, he thinks, may be obtained with time and pains without resorting to the open method.

The remarks of Mikulicz in December 1902, made in London as a tribute to Lord Lister, pretty well represent the German practice. He relates forty-five cases in twelve years in his own practice, of which thirty were operated on, with suppuration in four cases. In eight out of twelve cases X-rayed, the picture showed incomplete restoration in the anatomical sense, although to the touch bony union seemed to exist.

Bull said : " It is sufficient to know that death and disaster from amputation and suppuration and ankylosis have followed its performance in the hands of the many, and that we have no evidence that, when in the hands of the few it has proved safe, the ultimate results have been better than those by non-operative methods." With this last statement I in no way agree.

Dennis thinks that the future practice of the surgery of this and other countries will soon enable us to condemn it as an unsafe and unjustifiable procedure, or else it will be conceded as one of the grandest triumphs of our art.

Now these were the opinions of leading surgeons three years ago.

I know that at King's College, where Lister had shown the way, the open method has been a routine practice for many years; but not very long ago the professor of surgery there said to me, "It is a good operation, but there are very few surgeons I would allow to wire *my* patella."

There are those who argue that the ultimate results are as good with early movement and medico-massage as by the open method, and certainly the reports of eleven cases by Zum Busch are remarkable. Following on the Dutch, or method of Tilanus, he allows his patients to get up and go about in a simple splint on the second day, trusting to fibrous union, and thus preventing the adhesions which of necessity follow the complete fixation of the knee-joint. I do not believe that any results are as good as those obtained by the open method. You can never remove the effused blood, which is always present when the patella is broken, by any other operation. If there were no risk of suppuration, there is no doubt the open would become the recognised procedure. I hope to show that it is a fallacy to say that the results of medico-massage, etc., are as good. As medical referee, I see a fair number of cases which have been treated in the non-operative way, and I have had to report on several occasions that a man was quite unable to go up and down a ladder, or to do active work in a ship's hold, as long as eighteen months after his accident. As a rule, a working man is quite willing to take a little risk in order to get back quickly to his work, and prevent his home from being broken up. Now, what is this risk to-day? I do not think very great, even in cases not very carefully selected. One of my cases was a brewer's drayman, and another was a man in bad condition from syphilitic paralysis, and yet no trouble resulted. If trouble did result, I should make a very early opening on each side of the patella, and drain the joint behind the wired bone. If suppuration is prevented from getting into the pouches of synovial membrane or among the ligaments, the danger is not great. A case of compound

fractured patella which, on application at the out-patient room was found to be suppurating, was so treated by my assistant, Mr Litler Jones, and admitted to the wards under my care, and I watched the patient make a good recovery with a movable joint.

I have opened a great many knee-joints for conditions other than fractured patellæ, and I have no longer that terrible dread which one used to have. It must be admitted that if you have one disaster even in a hundred cases, the advisability of your practising the open method should be considered.

A practitioner in my neighbourhood told me that in his practice a case occurred in which the patient not only nearly lost her life, but in which the knee-joint became disorganised, necessitating amputation of the thigh, and subsequently her shoulder-joint had to be excised for a suppurative arthritis.

Ninety-one different methods of bringing the fragments together have been described, but with the exception of those of Dr Butcher of Liverpool, Mr Mayo Robson, and Mr Ramsay of Bournemouth, all are applied in a vertical direction.

The following are the main features of the method adopted in fifteen out of the sixteen cases put before you to-night. It is not desirable to operate at once, as by waiting a few days time is given to properly prepare the skin. I have described elsewhere the details of the preparation of the skin, etc., but I would say in passing that finding Kelly's method made my hands very rough and was very messy, I have used a turpentine mixture for the last two years with better results.

The formula is: Green soap 6 parts, glycerin 3 parts. Mix and boil, then add one-third turpentine.

For very hairy or very dirty skins, "Haarof," which is a sulphide of barium, and is sold for removing hair and wool from the lower animals previous to inoculation, is of great use. It does not irritate, especially if it is mixed with a little oxide of zinc.

It removes the hard dead epithelium over the ligamentum patellæ much better than a razor will. I now use it for all operations, especially emergencies. After thorough cleansing with the turpentine mixture, the hands are sterilised in alcohol and iodide of mercury. Freshly boiled silk is used for ligatures. Nothing is handled by any one except myself and *one* assistant, and both of us now use gloves, although for the first

ten of the cases my assistant alone used them. I thought the drill might tear the gloves, but I find by holding it in a sterilised towel that such a result is prevented.

A flap is rapidly turned up, and it is not necessary or

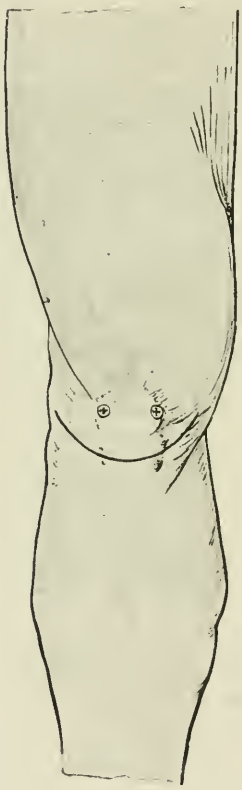


FIG. 1.—Shows best incision for avoiding pouching of the skin. The flap should not be carried above the middle of the upper fragment.

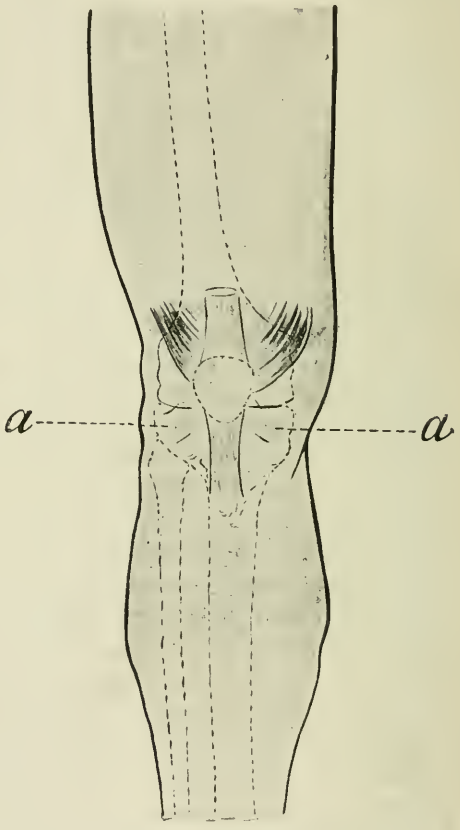


FIG. 2.—*aa*, Parapatellar ligaments attached to head of tibia and head of fibula, and which limit the lateral movements of the patella.

advisable to carry this above the middle of the upper fragment, thus avoiding any pouching of skin; the knife is then placed between the fragments so as to open the knee-joint (Fig. 1).

A considerable quantity of old blood clot, which is always

present, is removed with sterilised gauze. The fragments are freshened and trimmed with the knife only. Flushing of the joint has never been practised, and I think it almost reprehensible to waterlog the delicate vascular fringes with carbolic, perchloride, or other lotions.

The mechanical drill of Down is invaluable. It is used, with only a little freshening and adjusting of fragments, to go transversely and superficially through soft and hard structures, after which medium silver wire is placed by means of the cannula. It is not necessary to handle the fragments at all ; they are simply steadied while the mechanical drill goes through. The whole operation, including the suturing of skin, takes about sixteen minutes.

In one case only has the silver wire sprung, and this patient has bony union, and last week was engaged all day carrying 1 cwt. of coal on his back up seventy steps.

The advantages of the transverse method are that, when the wire is twisted it acts like a *splint* to the fragments, and compresses them very tightly, thus preventing any oozing of blood into the joint. It can be inserted quite superficially, and although there has been no necessity in this series of cases to do so, it could easily be removed by making a one-inch incision over each end of the wire. Finally, no trouble is caused from it in kneeling.

Catgut, silk, kangaroo and reindeer tendon have not been found satisfactory in the hands of various surgeons. Medium silver wire has been used throughout in my series of cases, a couple of silk sutures bringing the parapatellar ligamentous tissue over the front of the united fragments (Fig. 2).

In one case, out of the fifteen mentioned, the patella was in seven pieces, and an encircling wire had to be used.

The analysis of the cases speaks for itself. The charts show that after the first day the temperature has been uniformly normal. There has not been a single case of sepsis, and in one case only was there a little trouble around one stitch, not sufficient to give a rise of temperature, and due, I think, to the fact that the man had varicose eczema of the limb below and eczema of the scrotum and upper part of the thigh.

I have brought with me two patients—one a lad who has had both patellæ wired, and who runs along the roof-tops, in following his employment as a plumber ; the other a man who

TABLE.—*Analysis of Sixteen Cases of Fractured Patella Wired by the Transverse Method.*

No.	Sex and Age.	Employment.	Date of Accident.	Accident.	Date of Operation.	Allowed to move Knee-Joint.	Nature of Injury.	Progress after Operation.	Result.
1	M., 29.	Carter.	April 29, 1903.	Fell off a coal cart on to right knee. Knee-joint much swollen and hot.	May 11, 1903.	May 19, 1903.	Transverse fracture.	Without suppuration.	X-rayed March 3, 1905, bony union, wire broken, been carrying heavy weights, bends to right angle.
2	M., 41.	Brewer's drayman.	Dec. 2, 1903.	Slipped in ballroom and fell in sitting position — felt his knee give.	Dec. 10, 1903.	Dec. 27, 1903.	Transverse fracture — a good deal of loose tissue between the fragments.	,, ,,	X-rayed March 1905, bony union, wire in position, bends to right angle.
3	M., 26.	Acrobat.	April 27, 1904.	Slipped, jumping — felt his knee strike ground, and give, while trying to save himself.	May 9, 1904.	May 28, 1904.	Transverse fracture—large piece of tissue covered with blood clots bulged into knee-joint. Much organised blood.	,, ,,	Bony union, wire in position. Following employment as acrobat.
4	M., 24.	Carter.	Sept. 3, 1904.	Violent twist at football.	Sept. 12, 1904.	Sept. 30, 1904.	Transverse.	,, ,,	X-rayed March 1905. bony union, bends to right angle.
5	M., 15.	Plumber.	Sept. 17, 1904.	Fell off scaffolding on to both knees. Fractured both patellæ.	Sept. 26, 1904.	Oct. 24, 1904.	Right was in three pieces.	,, ,,	X-rayed February 1905, bony union. Follows usual employment.
6	M., 15.	Plumber.	Sept. 17, 1904.	,, ,,	Oct. 3, 1904.		Left—a piece of bone was turned into joint and had to be removed.	,, ,,	
7	M., 43.	Labourer.	Oct. 17, 1904.	Patient slipped, and in attempting to recover himself heard a "crack."	Oct. 24, 1904.	Nov. 6, 1904.	Right knee-joint inflamed and tender and full of fluid. Transverse.	,, ,,	X-rayed March 1905, bony union, wire in its place, bends to right angle. Carries hods of bricks.

8	M., 28.	Ship's steward.	Oct. 23, 1904.	Fell and struck left knee against thick iron stanchion. Suffered from syphilitic paralysis.	Dec. 13, 1904.	Jan. 2, 1905.	Operation seven weeks after — firm adhesions between patella and end of femur.	,,	,,	Bony union, good useful limb, wire in position.
9	M., 35.	Carter.	Nov. 18, 1904.	Horse kicked him on left patella — compound (wound corresponded to back part of horse-shoe).	Dec. 2, 1904.	Dec. 22, 1904.	Stellate (seven pieces), bradawl used through tendon of quadriceps and ligamentum patellæ, wire passed around fragments. Two silk sutures over top.	,,	,,	Bends to nearly right angle, bony union, wire seen surrounding patella.
10	F., 41.	Housewife.	Dec. 17, 1904.	Foot caught — fell down three stairs, and heard crack before her knee touched the ground.	Dec. 30, 1904.	Jan. 12, 1905.	Transverse. Much hæmorrhage into tissues. Organised blood in knee-joint. Two silk sutures over top of patella.	,,	,,	Bony union, bends to nearly right angle, wire in position.
11	F., 23.	Housemaid.	April 26, 1905.	Fell down the cellar steps.	May 1, 1905.	May 11, 1905.	Transverse fracture, two fragments.	First intention.		Bony union, moves to acute angle.
12	M., 23.	Farmer.	Aug. 20, 1905.	Fell from a loft, 10 feet high, on to his knee.	Sept. 5, 1905.	Sept. 15, 1905.	Three fragments, tarry blood in joint and small splinter of bone.	,,	,,	Bony union, moves to acute angle. Working as a carter on farm.
13	M., 49. (Mulcoy)	Painter.	Oct. 3, 1905.	Endeavouring to kick an offending dog, which he missed. Right patella.	Oct. 10, 1905.	Oct. 29, 1905.	Transverse fracture, three fragments, ¼-in. separation.	,,	,,	(Left was fractured nine years ago, not wired, wore a splint for many months.) Says right is as good at one month as left at eighteen months.
14	M., 64.	Tobacconist.	Oct. 13, 1905.	Fell off tramcar on to his knee.	Oct. 24, 1905.	Nov. 4, 1905.	Transverse fracture, two fragments, ½-in. separation.	,,	,,	(Bad vessels, wound healed up.) Patient died of apoplexy.
15	M.	Coal carter.	Nov. 22, 1905.	Fell off cart.	Dec. 1, 1905.	Dec. 20, 1905.	Three fragments, eye-ball suppurating, operation postponed.	,,	,,	Bony union.
16	M.	Fire brigade officer.	Dec. 2, 1905.	Motor fire engine ran into wall while he sat on it.	Dec. 2, 1905.	Dec. 14, 1905.	Compound, many fragments.	,,	,,	Superficial stitch trouble, probably auto-infection.

nine years ago had one patella treated by plaster, and who wore a Thomas' splint afterwards for eighteen months. He was supposed to have bony union, but you will notice that there is now three-quarters of an inch of separation between the fragments. His other patella I wired recently, and he tells me that he could do more with the wired one in a month than he could with the unwired one in eighteen months. The time in hospital need not be so long as is shown in my earlier cases, and I now take off the MacIntyre splint in ten days, allow them out of bed in a fortnight, and encourage them to move the knee, which they do to a right angle, in another fortnight.

I have had a number of old *un*wired fractured patellas X-rayed, and hope to show you what an enormous amount of separation takes place in some cases years after the patient has been lost sight of, even although a splint has been worn for many months.

This is a detailed account of my work on this subject during the last two and a half years, and if I have appeared to dogmatise in the presence of some of my old teachers, I hope they will understand that it is because I am convinced that this operation can now be done with safety, and that any success I have attained has been in great measure due to the early training which I received at their hands.

DESCRIPTION OF PLATES I.-III.

PLATE I.—Wiring by transverse method; shows neat way in which wires lie.

PLATE II.—Injury to an 18-year-old acrobat's knee, mistaken by medical man for fractured patella. Joint full of blood at once shows how tubercle and upper epiphysis are developed from same centre.

PLATE III.—The condition often found where the patella has not been wired. This man wore a splint for six months after leaving hospital (accident fifteen years ago).

2. THE RELATION OF ANGINA PECTORIS AND ALLIED CONDITIONS TO AN ARTERIO-CARDIAC REFLEX HAVING ITS ORIGIN IN THE ABDOMEN

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INTRODUCTORY.

ANGINA PECTORIS has attracted the attention, and at times riveted the thought, of many great clinicians during the past 130 years. That it should exercise the same influence over

Plate I.—To Illustrate Mr George G. Hamilton's Article.

PLATE II.—TO ILLUSTRATE MR GEORGE G. HAMILTON'S ARTICLE.

PLATE III.—TO ILLUSTRATE MR GEORGE G. HAMILTON'S ARTICLE.

the clinicians of to-day need not, therefore, cause surprise. Gibson (1) of Edinburgh, Oliver (2) of Newcastle-upon-Tyne, and Mackenzie (3) of Burnley have all three shown quite recently that they were the victims of the fascination or attractiveness of the subject. It requires no special courage to avow oneself a fellow-victim, however much courage it may require to attempt the elucidation of a subject which has been dealt with by so many able physicians of the present and the past. Were apology necessary for so doing, I would say that I have found the view which I am to submit of much practical value; that, instead of conflicting with past observations, this view interprets them, and that it gives the clue for the disentanglement of the confused mass of opinion on the subject. My views have emerged from the special attention which I have long given to cardiac and vascular disturbances, and more recently from a fuller knowledge of the processes of primary and secondary digestion, of nutrition, and of elimination. My indebtedness to the work of Pawlow (4), Herter (5), Chittenden (6), and others I gratefully acknowledge. The relations between gastro-intestinal conditions and vasculo-cardiac disturbances and the influence of dietetic substances upon the vasculo-cardiac system have recently been more and more impressed upon me, and this communication contains the conclusions at which I have arrived and indicates the path by which I have reached them.

Historical and Critical.

Heberden to Trousseau, 1768-1862.

We do not require to go further back in our historical retrospect than the date when the term "angina pectoris" was introduced into medical nomenclature. Those who wish to examine the earliest records of the symptoms called by this name will find the references in several of the older authors, particularly in Parry's monograph.

Angina pectoris was the name given by William Heberden (7) in the year 1768 to a disorder of the breast, which he thus described: "Those who are affected with it are seized while they are walking, and more particularly when they walk soon after eating, with a painful and most disagreeable sensation in the breast, which seems as if it would take their life away if it were to increase or to continue." "The seat of it and sense

of strangling and anxiety with which it is attended may make it not improperly be called angina pectoris."

As regards the nature of the disease to which he gave this name, he says that "the opinion of its being a convulsion of the part affected will readily present itself to any one" (p. 64).

In a later paper, of date 1785, containing the account of the *post-mortem* examination on the historical "unknown," he reports that there was no lesion found; and in his comments on treatment he advocated the use of medicines which "relieve and quiet convulsive motions" (vol. iii. p. 10).

Here at the fountain-head is found the idea of the nervous nature of the clinical phenomena of angina pectoris, a conception of the condition shared by many subsequent writers.

The next step worthy of note was made by Dr Caleb Hillier Parry (8), a physician in Bath, who published in 1799 a monograph entitled "An Inquiry into the Symptoms and Causes of the Syncope Anginosa, commonly called Angina Pectoris." This work is characterised by remarkable clinical acumen, and Parry's views, it seems to me, have not always been accurately presented by subsequent writers. Some of the clinical points dealt with by him are to be specially noted. He points out, for instance, that the first symptom is "an uneasy sensation (described variously as a stricture, an anxiety, or a pain) about the middle of the sternum, across the left breast, and in certain stages of the disorder usually stretching into the left arm" (p. 42), also that the pain occurs in paroxysms, and that early seizures seldom occurred without apparent cause (p. 43). He also insists, "we shall always find it (the pulse) become more or less feeble according to the violence of the paroxysm" (p. 45). Here we have an early recognition of great differences in the degree of severity in the leading symptom. The pathology of the condition was held by him, and by his friend Jenner, to be disease of the coronary arteries interfering with the blood supply to the heart. His induction was that a heart with such coronary arteries could not bear any extra strain put upon it, that if subjected to extra strain it became surcharged with blood, that its action became enfeebled, and might cease. The extra strain could be induced by mental emotion, by physical effort, and by gastro-intestinal conditions. As the heart condition advanced, even slighter causes than these might produce paroxysms. Thus heart failure and its organic cause formed the central, and to him the essential, factors, in determining the gravity of the condition. According to Cullen's nosology this was assuredly a *syncope*. "The motion of the heart diminished, or even for some time ceasing. *Motus cordis imminutus, vel aliquamdiu quiescens.*" It differed from an ordinary syncope "only in being preceded by an unusual degree of anxiety or pain in the region of the

heart." He therefore termed the disorder *syncope anginosa*. To make the position of this old Bath physician still more clear let me give his own words regarding a patient who had been long subject to attacks of what he considered a "very pure angina pectoris." He accompanied his patient on a walk up hill in order to witness what occurred during the fit. He says "when the paroxysm was thus excited, I could perceive no symptom of disorder in addition to the uneasiness in the breast, except a gradual and most evident diminution in the strength of the pulse" (p. 59).

Allan Burns (9), Professor of Anatomy and Surgery in Glasgow, and practising in that city (1809), accepts Parry's view that syncope anginosa originates "from some organic lesion of the nutrient vessels of the heart" (p. 137). He compares a heart with "cartilaginous or ossified" coronary vessels to a limb with a ligature round it called into active exercise. The limb so bound can only work for a very short time; "it soon fails and sinks into a state of quiescence" (p. 138). He holds that "the essence of this complaint consists in a reduction of the action of the heart" (p. 146).

In 1815 Jurine (10), in an essay to which the Academy of Medicine awarded a medal, combated Jenner's and Parry's view that angina pectoris was due to ossification of the coronary arteries. To him "sans douleur sternale, il n'y a pas d'angine de poitrine" (p. 72), and he argued that the angina was essentially due to an affection of the pulmonary nerves (p. 123).

Laënnec (11), in 1819, had found that in several subjects who had laboured under angina pectoris, and in whom there co-existed either hypertrophy or dilatation of the heart, there was no ossification of the coronary arteries (p. 757). According to him, "angina pectoris in a slight or middling degree is extremely common, and exists very frequently in persons who have no organic affection of the heart or large vessels" (p. 756). Desportes (1811) is quoted by Laënnec as holding similar views. Angina pectoris was to Laënnec a neuralgia.

Forbes (12) (1832), the translator into English of Laënnec's great work, divided cases of angina pectoris into organic and functional. The pain to him was the result of "some unknown temporary condition of the nerves of the part," and he believed that "such a morbid condition of the nerves may be produced in a heart in all other respects sound."

Bertin (13) (1833) also repudiated the view that angina pectoris was due to ossification of the coronary arteries. He had seen the symptoms "accompanied with a multitude of organic affections, either of the heart or of the large vessels, or even of the lungs" (p. 410).

Hope (14) (1839) held that "the nature and variability of the

symptoms of angina pectoris confirm the opinion of Laënnec that it is a neuralgic affection" (p. 499).

Latham (15) (1846), along with a vivid portrayal of angina pectoris, considered the disease to be "a certain assemblage of symptoms, and not any constant pathological condition belonging to the structure of the organ" (p. 399). The paroxysm was to him "plainly a compound of pain and of something else" (p. 385). It was a "spasm of the heart," as it had been to Heberden. But then note his conception of spasm. He says: "Spasm is always accompanied with pain. And pain and spasm, wherever they are, disable the part which they befall" (p. 385). "In spasms of smaller degree the heart fails to close freely upon the blood and to impel it freely into the arteries. In its spasm of greater degree it fails to project it altogether" (p. 386).

Walshe (16) (1851) looked upon angina pectoris as a "paroxysmal neurosis in which the heart is essentially concerned" (p. 190). He thought genuine angina pectoris to be a very rare disease, while on the other hand he "very frequently met with a form of complaint combining in a minor degree many of the characters of angina." To this "imitation of the true disease" he gave the name of "pseudo-angina." He thought that this was the explanation of Laënnec's notion that the condition was of very frequent occurrence (pp. 203-4).

Stokes (17) (1854) agreed with Parry, and says "that the symptoms of angina arise from a temporary increase of weakness in an organ already weakened" (p. 486). He disputes Latham's idea of spasm, and thinks the idea of syncope more in accordance with observed fact.

Trousseau (18) refers to the variety of pathological change which had been described as having been present in the hearts of persons who had suffered from angina pectoris. On the other hand, he has of necessity to admit that angina pectoris, even when most intense, need not be a symptom of an organic lesion. He therefore regarded the organic changes, when they were present, as non-essential, as coincidences, and held that the real affection was a neurosis—in fact a neuralgia. That there might be no ambiguity in the minds of his hearers he again repeated his opinion that it might be only "an idiopathic neuralgia."

Three Views.

At this point we may draw a line and say that up to it there were, if we exclude Jurine's view—which we may safely do—three views regarding angina pectoris: the first, that it was a convulsion of the heart, or a spasm of it; the second, that it was due to interference with its blood supply; and the

third, that it was a neurosis—the word having no subtle or obscure significance, but meaning, simply and plainly, a neuralgia—an idiopathic pain in nerves. Up to this time, it is also important to note that the new term was not limited to a relatively rare malady, characterised by a few dramatic incidents and a suddenly tragic conclusion. Its use had become so extended that Walshe deemed it desirable to introduce the term pseudo-angina, under which to range the cases that, in spite of the correspondence in symptoms, had not the grave significance that the fatal angina of Heberden presented.

Angina Pectoris Vasomotoria.

With the year 1867 a new factor was introduced into the discussion on the nature of angina pectoris. Nothnagel (19) in that year wrote a paper which he entitled "Angina Pectoris Vasomotoria." In this condition the symptoms closely resembled those of angina pectoris, and would certainly, I think, have been placed under that designation by some of the distinguished clinicians whose works have been referred to. The essential point in this paper was the recognition of the important clinical fact that in this group of cases narrowing of the arteries preceded and caused the cardiac embarrassment and other symptoms; that, in fact, the anginous symptoms were secondary to the vasomotor ones; and that by relieving the cramp in the vessels the anginous symptoms were prevented coming on.

Earlier Observations on Blood Supply and Muscle Pain.

Before Nothnagel, Latham, when dealing with the treatment of persons the victims of angina pectoris, had not failed to note the guidance to be obtained from a true estimate of the condition of the vascular system. He says:—

"The paroxysm is often put off and its severity mitigated and life prolonged by no means more surely than by keeping the vascular system in a just balance between fulness and emptiness, between rich blood and poor blood" (p. 405).

Three years before the date of the publication of Latham's lectures, Sir Benjamin C. Brodie's "Lectures on Pathology and Surgery" (20) were published (1846). In them there is a lecture on senile gangrene which I doubt if any living surgeon could

add to. He notes that the condition might be due to ossification of arteries or to obliteration without ossification. He enlarges upon the prodromal symptoms of gangrene—he had noted the numbness, pain, and loss of muscular power—which may precede its visible manifestation. With the clinical instinct of the great men of his time he applied these observations to interpret the much discussed question as to the nature and causes of the pain in angina. The paroxysm of pain in angina varied in intensity, so it did in gangrene, greatly. The pain in gangrene might become excruciating—the pain of angina might fail to find words adequate to convey a true sense of its anguish. "The disease," he says, "is called neuralgia, which," he adds, "means nothing" (p. 356). A clear thinker, as well as a masterly observer, this surgeon of sixty years ago!

A still earlier recorded observation bearing upon the relation between lessened blood supply and pain must not be omitted were it only for the sake of historical accuracy. At the Académie Royale de Médecine, the *Séance* of October 4th, 1831, Boullay (21) submitted an observation he had made on a mare of a condition to which he gave the name of "intermittent claudication." The symptoms came on brusquely after some minutes' exercise, and they were found to be due to obliteration of the femoral arteries. When the animal was in repose the blood was able to reach the limbs by the collaterals, but when it trotted the compressed collaterals did not furnish enough blood, and the limbs were seized by *engourdissement* and by severe pain which made the animal fall.

Charcot (22) twenty-six years later revived this subject, and drew attention to a corresponding condition as it occurred in the limbs in man as causing severe pain, and as a premonitory sign indicative of a tendency to senile gangrene. So far as I have found, he did not apply his observations to the elucidation of the symptoms of angina pectoris as the English surgeon Brodie had done ten years earlier.

After this, however, intermittent claudication as a cause of painful muscle spasm took its place in medical literature, and has been much used by later writers when discussing the cause of the pain in anginous paroxysms.

Arterial Spasm.

Standing alone, and forming an epoch by itself, is Lauder Brunton's (23) discovery (1867) that dilatation of the peripheral vessels by the inhalation of nitrite of amyl removed or relieved anginous symptoms. From this date vasomotor spasm became a still more definite factor in the consideration and the interpretation of the "syndrome of symptoms" known as angina pectoris.

What is included under "Angina Pectoris"?

What, then, are we to regard as angina pectoris? Are we to confine the use of the term to that type of case, described with such dramatic force by some of the earlier writers, that there still lingers round its earlier records a glamour of picturesqueness? Are its symptoms to be bounded by the *dolor pectoris* and the *angor animi* of its early recognition, and is its close to be characterised by the same tragic suddenness? I think not. From the time of Parry the term "angina pectoris" has been applied to symptoms of less gravity, and to conditions where the outlook was not necessarily grave. Let me clinch this statement by quoting the words of Professor W. T. Gairdner (24), who, writing in 1877, says :—

We now know that this typical angina is only the culminating form of a group of symptoms, which in their less pronounced, less definitely painful, and more complicated forms, are found to permeate the whole field of cardiac pathology and diagnosis (p. 570).

From his own personal experience he says :—

There is often an element of subjective abnormal sensation present in cardiac diseases which, when it is not localised through the coincidence of pain, is a specially indefinable and undescribable sensation (p. 565).

A sensation which can only be called anxiety or cardiac oppression (p. 566).

To this group of symptoms he gave the special title *angina sine dolore*, recognising thereby what he believed to be "its true diagnostic and pathological significance and its alliance with the painful angina of Heberden." Even the dolor pectoris is, then, no longer an essential symptom; and the angor animi is whittled down to a sensation of anxiety. This shrewd clinical pathologist saw, however, that there were phenomena which, while different to, were of the same kind as those of the classical angina. This is what Walshe recognised when he used the much-abused term, "pseudo-angina." While Gairdner places angina amongst the neuroses he hardly, I think, attaches the same meaning to the word as the earlier writers did; and I am not sure that he does not somewhat grudgingly stick to

the term. He was clearly impressed by the possible importance of vasomotor spasm.

Sansom (25) on the other hand (1892) thinks it probable that a paroxysm of angina pectoris is an intense nerve storm, provoked by impulses conveyed to certain cerebro-spinal centres (p. 13). He refuses to adopt the theory of cardio-arterial spasm as the indispensable cause of the pain (p. 438).

Osler (26), in his erudite lectures on the subject (1897), after defining angina pectoris, proceeds to say that the term is employed generically to define paroxysmal attacks of pain in the chest, qualifying the varieties by such names as true, false, hysterical, and vasomotor (p. 8).

Douglas Powell (27), in a very admirable article, classifies angina pectoris under (1) a vasomotor group, and (2) angina pectoris gravior, which includes secondary and primary cardiac angina. Forbes (12), Bramwell (28), Gibson (29), Oliver (2), and others recognise two classes, organic and functional. Huchard (35) insists that there is only one angina pectoris, and that it is always due to ischæmia of the heart muscle.

From all this it is quite clear that the term is no longer confined to the severe and fatal malady described by Heberden. It has been extended to a great variety of conditions, characterised by some sense of precordial discomfort, or of discomfort referred directly to the heart, which may or may not be accompanied by pain, and which may or may not be associated with coarse anatomical lesion. It was to cover these lesser manifestations that such terms as pseudo-angina, angina notha, false angina, and functional, as distinct from organic, have been used and applied.

Angina is indeed, to use Osler's words, "a syndrome or symptom group," the clinical pathology of which has to be determined in each separate instance. The term angina pectoris has in fact the same nosological significance as the term dropsy formerly had. It is not a disease; it is a symptom or a group of symptoms, and it is no longer the symptom but the cause behind and determining it which is the problem presented to the clinical pathologist of to-day.

Constructive and Critical.

In turning to the constructive side of this communication I ask you to look with me at certain vasomotor phenomena which are to be observed in the body; and I hope to make it

clear what I regard to be the relations of those phenomena to the phenomena of angina pectoris.

The presence of arterial spasm has been recognised by many who have written on angina pectoris since the date of Lauder Brunton's epoch-making discovery. Views as to the causation of the spasm have been various, and the relation of the spasm to the symptoms has also been variously interpreted.

An Arterial Abdominal Reflex.

I ask you to consider with me certain observations regarding an arterial reflex having its origin in the abdomen, the importance of which, I think, has not hitherto been fully appreciated. After taking food, and for at least part of the time during which the active processes of digestion are under way, there is an influx of blood to the whole splanchnic area, leading to hyperæmia, such a hyperæmia as considerably reduces the volume of blood in parts outside this area. This drainage into the abdominal vessels is balanced in the general circulation by a systemic arterial contraction. This is evidently a reflex phenomenon originating in the splanchnic system, passing to the vasomotor centre in the medulla, and thence transmitted to the systemic arteries. The existence of this set of phenomena has been carefully worked out by Dr George Oliver (30) in the average healthy person. The arterial contraction is shown by a definite diminution in the diameter of the vessels. It is to be regarded as a physiological hypertonic contraction, which in the average person is accompanied by a rise of blood pressure. However that may be, there is no doubt about arterial narrowing; and I am content to build upon this one certain and positive fact. The fact, then, with which I begin is the change in the systemic arteries during active digestion—whether it be called narrowing, contraction, or hypertonus, signifies not, so long as the word we use expresses our thought. The change will be found to be a reduction in size and an apparent thickening of the arterial wall. The degree of these arterial changes depends upon the kind of meal which has been taken. In the big eater and the wine-drinker the arterial contraction is associated with a rise of blood pressure and a true increase of arterial tension.

Putting extreme cases aside, the general statement will be found to be correct that narrowing of the arteries is present,

but in many people, both middle-aged and old, I do not believe that there is any constant rise of blood pressure along with it. To the educated finger and the analytical mind there is no doubt about this, whatever our instruments show. The point is not a purely academic one, for the small and constricted vessel, with its slight systolic expansion, is very commonly regarded as an indication of heart debility and treated as such. In other cases the mistaking of hypertonus for a rise of blood pressure may be of no practical consequence, for the treatment applicable to the one is suitable for the other.

The point here insisted on is the existence of this arrangement for adapting the circulation to the normal processes of ingestion and digestion of food. It is a normal reflex process. It is a systemic vessel constriction originating in a normal stimulus in the abdomen, and brought about through the anatomical connections in the nervous system. It is not, therefore, a "neurosis"; nor is the exaggeration of this reflex, prompted by a large meal of stimulating foods, a neurosis. It is necessary to lay emphasis on this distinction. This reflex, like all normal reflexes, is a beneficent arrangement, and could be easily shown to be one of the means of protecting the body from injury. It varies in delicacy in different persons; it exists in all.

Exaggeration of Normal Abdominal Reflex.—Granting the existence of this normal reflex, it will not be questioned that high feeding and alcoholic liquors accentuate it; and that under such circumstances there is marked raising of arterial tension in addition to arterial contraction.

It must, however, be here recognised that in the latter condition there are two elements present—one, the vascular reflex; and second, the passage into the circulation of substances produced in the processes of digestion, and of substances derived from the alcoholic liquors drunk. It is not possible at present to give these two factors their precise place in bringing about the accentuation of vessel constriction and of stimulating the increased tension. The existence of the reflex is granted, and there is really little room for doubt that certain substances present in the blood can directly stimulate vessel contraction. Contraction so induced is not a neurosis; it may, indeed, be induced when the connection with nerve centres is destroyed.

Action of Reflex on Sclerosed Arteries.—This recurring arterial contraction, especially if associated with increased arterial tension, is, as I pointed out some time ago (31), the efficient cause, and the usual cause, of arterio-sclerosis in the sense in which this term ought to be used (32). At the same time I dwelt upon the importance of recognising that sclerosed vessels are not, because of the change in their walls, incapable of contraction, but that, on the contrary, they are prone to become hypertonic from causes which are commonly regarded as trifling. Professor Pal (33), as seen in his recently published work, "Gefässkrisen," has also observed that sclerosed vessels are particularly sensitive, and specially liable to hypertonic contraction.

Let us bear in mind then, *first*, the existence of this systemic arterial reflex having its origin in the digestive organs or processes. Secondly, the influence of the character of the material introduced into the digestive system in accentuating the reflex. Thirdly, the effect of the reflex in producing arterio-sclerosis. Fourthly, that sclerosed vessels are particularly liable to hypertonic contraction.

Relation of Reflex to Angina Pectoris.

The relation between the phenomena just dealt with and angina pectoris is not at first sight apparent; and, in order to demonstrate the close connection between the two, I must now as briefly as possible refer to several illustrative cases which I have recently observed.

Case I.—*Angina Pectoris Gravior, showing Extreme Sensitiveness of Arterio-cardiac Reflex and its Production by taking Nourishment.*

The patient was a professional man, aged 67, whom I saw for the first time on September 6th, 1904. He had suffered for years from definite and characteristic attacks of angina pectoris. Lately he had been much worse, and had been confined to bed for weeks. When I first saw him he was having many attacks daily, and circumstances of the most ordinary kind evidently brought them on—such circumstances as the taking of nourishment, the presence of flatulence, slight excitement, and even the changing of his position in bed. The

severity of the attacks varied within considerable limits; the more severe, and therefore the more typical, attacks began with pain in the left elbow, which soon extended upwards to the shoulder and then downwards over the precordia. During an attack the hands became cold and painful. The medical attendant, who was watching the patient with the greatest assiduity, informed me that the pulse always indicated the advent of an attack; the radial artery became more definite, firmer, and smaller, the pulse-rate increased, while the wave became small, and might almost disappear. In some of the worst attacks the patient had become unconscious and was thought to be dying. The treatment followed was the administration of nitrite of amyl and nitro-glycerine for the more pronounced of the attacks, and the relief afforded by them had always been prompt. Under the influence of one or other the pulse wave became larger, the vessel wall softer, the hands became warm, and the pain passed off. The patient took a remarkably calm and intelligent interest in his attacks, and the foregoing is gleaned from his own and the medical attendant's description. The diet for some time had consisted almost exclusively of raw eggs and weak broth, while sedatives of various kinds had been employed medicinally. The patient had always been a large flesh eater. There was no albumen or sugar in the urine. The medical attendant had been impressed by the fact that the patient more recently almost always had a seizure after being given nourishment, even when that consisted of some raw egg. I had the opportunity of confirming the observations on the change in the pulse during a paroxysm soon after my arrival at the patient's bedside. During the time I spent talking quietly to him the pulse was small, soft, and feeble. I examined the heart without causing any discomfort. The sounds were so faint that they were occasionally inaudible, although the chest wall was thin. I next proceeded to examine the abdomen, and had my hand placed quite lightly on the epigastrium when the patient told us that an attack was coming on. I at once removed my hand. He took his attack with remarkable composure, and it was evidently not accompanied by the distressing sensations which so frequently accompany anginal seizures. Pain began in the left elbow, which, as has already been noted, was always the premonitory symptom of the more severe seizures, and extended

to the shoulder and then to the precordia. The face became pale. The radial artery became quite abruptly hypertonic—that is, it became firmer and smaller, the rate rose to 100 and 110, and the tension was sensibly raised. He was at once given a tabloid of nitroglycerine, and in a very short time the artery relaxed and the pulse became large, strong, and rather thumping, the pallor of the face was exchanged for a ruddy tint, and the pain ceased. The thumping soon passed off, and the pulse resumed the small feeble character it had presented at the outset.

Comments.—There could, of course, be no doubt that the patient was the victim of a severe form of angina pectoris, and that the attacks were accompanied by marked arterial constriction. It was fortunately easy to arrest the seizures. They had, however, persisted for weeks without any lessening in their frequency or in their severity. The question which awaited solution was, Could anything be done to lessen the tendency to the attacks? Palliation was easily achieved; but could any measures be adopted which might be more curative? The point in the history which was most uncommon was the fact that the taking of nourishment was almost always followed by an attack, although the amount taken was small and could hardly be further reduced. Acting, however, on this indication, it was decided to materially alter the diet, the nature of which has been already stated. The dietary now advised was to consist of peptonised milk, peptonised oatmeal gruel, etc., and one raw egg well beaten up daily. The patient was moreover given small doses of arsenic, tincture of strophanthus, and spirit of nitrous ether. This line of treatment was persisted in, and when I saw the patient a fortnight later the report of his condition showed considerable and satisfactory improvement; the attacks had greatly diminished in number and severity, and the taking of nourishment was only occasionally followed by an attack; the pulse was stronger. The tongue, however, had become coated, and small doses of rhubarb and soda were advised.

A week later the report was that the attacks had still further lessened in severity and frequency, and that when an attack was threatened it could be averted by a dose of aromatic spirit of ammonia or even by a dose of rhubarb and soda

mixture. When pain in the elbow came on, it was accepted as an indication that the threatened attack would be somewhat severe, and the patient was at once given a small dose of nitrite of amyl. The sensations which now more ordinarily preceded an attack were a sense of discomfort and distension in the stomach, and attacks were often associated with definite flatulent distension, the escape of gas being followed by relief. The medical attendant noted that when the abdominal discomfort supervened the pulse rate increased, and there was distinct hypertonus ; as the attack passed, the vessel wall relaxed and the rate returned to normal. Within the next two months the patient's condition varied a good deal, but it was noted that any gastro-intestinal or digestive upset at once threatened to induce an increased number of attacks. The management of the diet and of the digestive system required much tact and judgment, and was admirably conducted by the doctor in charge. In some weeks the patient had improved so much that he was able to be out of bed and to move about the house for the greater part of each day, and to interest himself in outside affairs. His clinical history for the succeeding months was chequered, for as his condition improved, it became impossible to continuously diet him as was desirable ; still, at the end of June 1905, he had not had a seizure for six weeks, and was in fact freer of attacks than he had been for years.

Soon after this, however, albuminuria made its appearance with anasarca. The anasarca was got rid of, but the albuminuria persisted ; Cheyne-Stokes breathing developed and became extreme. The patient finally developed gangrene of his entire right lower limb, of which he died in the end of October 1905, a proof that his arterial system was more degenerate than the radials indicated. I submit the case as illustrating an extreme exaggeration of the normal reflex which has been described. The angina in this patient was associated with a weak and somewhat enlarged heart. The hypertonic spasm of systemic arterioles, resulting from gastro-intestinal stimulus, proved so severe a strain on the weakened heart as frequently to lead to a syncope, during the anginal paroxysm, which was only short of being fatal. If we had had the opportunity of seeing the heart, it is probable that atheromatous or ossified coronary arteries would have explained its debility. The instructive feature was the extraordinary sensitiveness of the peripheral

organs from which the reflex started, and the success in allaying that sensitiveness by changing the kind of nourishment given, and its partial predigestion. The remedial effect was so pronounced that it gave me the impression that in this patient the normal reflex had been irritated beyond control by the free use of the more pure proteid foods, until it responded to the most trifling local irritation. That the anginous symptoms were always determined by the vascular hypertonic contraction was undoubted, and, so long as the latter could be restrained, there was no angina, and, as has been indicated, the key of the position was in the alimentary system.

CASE II.—*Condition allied to Angina, showing the Stimulation of the Arterio-Cardiac Reflex by Digestive Disturbance.*

This case might be classed as angina sine dolore, but I use it as an example of conditions allied to angina pectoris, in which hypersensitiveness of the abdominal reflex gave rise to phenomena of considerable interest and importance.

The patient, aged 68, was a member of one of the learned professions, and had spent many years in the tropics. I saw him in November 1904, during convalescence from an influenzal broncho-pneumonia, and was informed that on the afternoon of the previous day, between four and five o'clock, he had had a "fainting attack," in which his face became pale, he broke out into a cold perspiration, and the pulse was imperceptible. The nurse had been greatly alarmed, as she thought the patient was dying. He soon, however, rallied from this, and I saw him in the afternoon of the following day. It is unnecessary to enter into the details of this case at this stage, beyond saying that when the medical attendant and I inquired fully into his dietary it was found to be very faulty, and he was forthwith put upon a comparatively austere regimen.

He had no similar attacks during the following days, but he often spoke of an attack as impending, and he apparently expected an attack about four o'clock in the afternoon. It was thought by his family and the nurse that this was pure nervousness, and an expression of the pessimistic view he tended to take of his condition. Five days later I again saw the patient

and obtained from the nurse the statements on which the preceding account is based. I was waiting by the patient's bedside for his medical attendant to arrive, and thought the patient, who was sitting propped up in bed, looking well, for his colour was good. His pulse was 80, soft, small, and feeble. The respiration, however, showed a long deep breath often with a little sigh, followed by several quiet inspirations; it was distinctly "cerebral" in type, and it made me somewhat anxious. I asked the nurse to give him half an ounce of whisky in water, although he had had a couple of drachms shortly before, as I wanted to see the effect it would have upon the pulse and respiration. The effect was unexpectedly striking and even alarming. In a few seconds the pulse altered greatly in character: the artery very plainly tightened up, becoming smaller and harder, and the rate varied within very wide limits. It was frequently 88 per minute, then it would run up to over 100, and again sometimes fall even to 40. Along with this sudden irregularity in the pulse rate there were equally pronounced respiratory and cerebral phenomena. Lying with his eyes shut the patient seemed to fall asleep, and the breathing was so quiet and shallow that only on close attention could it be seen that he was breathing at all. Then he wakened up with a start and a groan, followed by some deep breaths. This series of phenomena went on with perfect regularity as long as he was not spoken to, each part of the cycle only lasting some seconds. When asked on awakening what was the matter, he explained that he had become unconscious, and had been dreaming that he was falling down a precipice, or passing through some equally unpleasant experience, often in company with some old friend long dead. He spoke of these attacks as "delirium," and insisted that the present was only an exaggeration of what he had experienced every afternoon, and to which reference has already been made. He could be kept awake by speaking to him, and he preferred being spoken to, because, as he said, it helped him to fight against the attacks. When he woke up from the nightmare his words were sometimes incoherent and unintelligible for a few seconds, but he rapidly regained complete self-possession.

This alarming and curious condition lasted about an hour. It had come on about three hours after lunch, which had consisted of a chop, spinach, and one potato. Some time after

that he had had milk and hot water and two drachms of whisky in lieu of afternoon tea. When the patient's condition was as has been described, I asked the nurse to make some tea, and he was given two-thirds of a moderate-sized cupful of this. The attack had completely passed off by the time the doctor arrived ; the pulse had returned to the condition it was in before the attack, the breathing was regular, and he was quite calm and self-possessed. When the next meal came he was quite able to feed himself. I had no doubt the patient's view was correct that the symptoms shown this particular afternoon were only an exaggeration of the afternoon symptoms which he had spoken of, but which no one with more understanding than a nurse had witnessed. The doctor had called at various hours for the purpose of observing the attacks, but the stimulus of his visit appeared to be sufficient to postpone them.

Comments.—The phenomena in this case were very striking, and the symptoms could only be regarded as indicating an anxious condition of matters. The fact that they threatened chiefly in the afternoon, and had so far lasted only for a comparatively short time each day, was reassuring ; while the time of their occurrence suggested a connection between the symptoms and the stage of the digestion of the mid-day meal, which was the principal meal of the day. Suspicion was all the more strongly directed to this view by the fact, of which I had no doubt, that the attack I witnessed was precipitated or intensified by the half ounce of whisky given at my suggestion. The immediateness of the effect was startling: it gave the impression of being produced the moment the draught reached the stomach. It was indeed impossible to escape from the conviction that the phenomena pointed to the production by the alcohol of a peripheral gastric irritation or stimulation, which precipitated or intensified a condition which was already threatening. On this assumption the local irritation must have been followed by a very prompt reflex effect. The impulse communicated by the sensory nerves of the stomach to the vasomotor centre must have stimulated that centre so as to increase its vaso-constrictor action, the result being the hypertonic contraction of the radial artery which was so markedly present. This stimulation of the vasomotor centre must have also told in some way upon the cerebral blood supply, judging from the cerebral manifesta-

tions which were present. That even the motor area of the brain was involved was shown by the occurrence of muscular twitching and starting of the limbs, especially on the right side, when the other cerebral manifestations were at their maximum. The respiratory centre, as shown by the great respiratory irregularity, shared in the perturbation; this association, however, is of common occurrence. That the attack should have passed off as it did, without any therapeutic measures being taken beyond the administration of a few ounces of tea infusion, only strengthened the view that it had been determined by peripheral conditions of a temporary kind; that, indeed, the true cause of the trouble lay in an error in the digestive process at this particular period after the mid-day meal.

The diet was altered in some of its details, and for an afternoon or two the patient was given a dose of one of the commonly-used vaso-dilators as soon as the symptoms threatened to come on. He had no more attacks; his recovery was complete, and he has returned to his duties in the East.

CASE III.—*Anginous Paroxysms first appearing on Physical Effort: Influence of Emotion and of Diet.*

This patient was a public servant, whom I saw in July 1905. He had suffered for some time from attacks of typical angina pectoris, commencing with pain referred to the heart. This symptom first appeared when he was hunting, and so constantly reappeared when he attempted to follow the hounds that he had to stop doing so. He had had one or two attacks of great severity during the night, in at least one of which it was thought he was to die. He had been treated with considerable success by an eminent Irish physician. He carried about tabloids of trinitrin in his waistcoat pocket, and had been taking potassium iodide for some time. His condition had become practically stationary; hunting was out of the question; he could only walk on level ground; the slightest hill, or the slightest hurry, produced a breast pang, which arrested further activity. When I first saw him he was having a slight attack almost nightly, soon after midnight, and at that time he usually took a tabloid of trinitrin. He claimed to be

a small eater, and his daily wine consumption did not exceed two or three glasses. His dietetic habits had not been seriously inquired into, and had not been altered. He had been told that the artery of his heart was rigid, and he and his family were dwelling under the shadow of the fear of sudden death at any moment. On examining him I found that the heart was slightly enlarged, the dulness reaching to the nipple line, but the sounds were clear and pure at apex and base. The pulse was regular, of good strength, and gave no indication of an enfeebled myocardium. The vessel wall was not much thickened for a man of his age, and in view of the arduous life he had led at home and abroad ; there was no albumen or sugar in the urine, but it tended to be scanty and high-coloured. The bowels were acting, but not freely, and his conjunctivæ indicated " sluggish liver." The question here, as in Case I., was whether anything could be done to carry improvement beyond the point it had reached. I was again fortunate in witnessing a slight attack in this patient. When I was examining into the state of his heart and vessels, which under the circumstances I was doing with great care, and not hurriedly, he became a little agitated and informed me that he was having a slight attack, with the usual feeling in his heart. On putting my finger on the radial pulse it had become markedly hypertonic. In a very short time the hypertonic spasm relaxed, and the heart sensation vanished.

Here was another instance of the pain and anxiety of angina, assuredly due to arterial spasm. There seemed to me, further, no doubt that the measure of emotional disturbance aroused by the medical examination had induced the attack. There was, therefore, no doubt as to the existence of marked vasomotor sensitiveness. As the patient was already taking potassium iodide more or less regularly, and trinitrin when threatened with a paroxysm, I felt that not much benefit could be expected from further medicinal measures. Guided again, therefore, by the theory that this vasomotor sensitiveness owed its origin to the circumstances and conditions of the alimentary system, I strongly advised that all wine should be stopped, and that other changes be made in the diet. The progress of the case need not be enlarged upon ; it is sufficient to say that the patient made marked improvement in every respect.

CASE IV.—*Angina with Gastric Disorder: Attack caused by Palpation of Epigastrium.*

This was a female patient in the Royal Infirmary, who complained of attacks of pain in the chest, over the precordia, with shortness of breath and of indigestion and pain in the epigastrium. The heart was weak and somewhat dilated. I mention this case on account of an experience I had in connection with it somewhat resembling an incident noted in Case I. On examining the patient's abdomen I found that there was surface hyperæsthesia in the epigastrium, as is common in some forms of gastric disturbance; and as I was gently palpating the region, I noticed the patient's face become pale and expressive of anxiety and discomfort. I at once put my finger on the pulse, and asked her what was the matter; she replied that the pain in her chest had come on. The radial artery had become markedly contracted and hardened. As the hypertonic spasm of the artery relaxed the heart discomfort passed off.

CLINICAL PATHOLOGY.

We are now in a position by gathering up our separate points to explain the whole clinical pathology of angina pectoris. In the first place there may or may not be a morbid anatomy. When present, it varies so widely in different instances that, from the earliest times to the present, many physicians have refused to accept any anatomical change as sufficient to explain the phenomena. Whenever this is the state of knowledge it will be found that men turn to that scapegoat of all that is unknown— the nervous system.

In attempting to interpret the phenomena of angina pectoris, I shall begin with an illustration of the simplest form of angina. Tobacco poisoning, from smoking, is commonly recognised as the cause of a form of angina pectoris—a toxic angina. It so happens that I can give myself tobacco angina by smoking strong tobacco. The symptom which arouses me to the fact that I must put my pipe aside is a sense of constriction at the heart, a feeling as if it were grasped; it is not a very unpleasant sensation, but it gives one the impression that it only required to be much intensified to make the dolor pectoris to be avoided, whatever self-denial it required. The heart discomfort is accompanied by a hypertonic contraction of

the radial arteries. Discomfort and hypertonus disappear together. If I am told this is a neurosis, all I can reply is, if it is, opium poisoning, or tetanus, is also a neurosis ; and that I do not look at morbid processes from that standpoint.

Whatever the degree of intensity the heart-pang may reach, it is, I believe, always the result of a *sudden* embarrassment of the myocardium, and usually and chiefly that of the left ventricle. This sudden heart embarrassment, varying widely in degree and intensity, is the one fact common to all the divisions or subdivisions under which all cases have been classed. The angor animi, which also varies in degree, is, I think, common to all sudden heart embarrassments, which fall short of being so immediately fatal that there is no time for the development of such sensations.

This heart embarrassment is, as Mackenzie (3) has quite recently pointed out, an " impairment of the function of contractility." This is the modern expression of what Parry meant by " syncope." It is to this condition, associated with pain, that Rosenbach (34) confines the term " stenocardia."

The impaired contractility is due either, in the first place, to the sudden strain put upon a feeble myocardium by general arterial contraction, or by a call for increased work ; or, in the second place, to a myocardium feeble, not from anatomical changes in it, but temporarily so, from deficient blood-supply, the result of the participation of the coronary arteries in a general arterial spasm.

The cause of the sudden heart embarrassment in the vast majority of cases is the arterial hypertonic contraction, or spasm contraction, we have been considering, the latter term indicating the more sudden and severe degrees of contraction. Even toxic conditions only give rise to angina when the factors indicated are present.

By means of the cases reported I have endeavoured to illustrate the remarkable relations which exist between the digestive system, or the materials introduced into it, and the general arterial system. I have also shown that conditions having their origin in the abdomen can induce a hypersensitiveness of a normal vasomotor reflex, which becomes apparent as arterial spasm, whenever the exciting conditions are sufficiently pronounced ; and that when those conditions are altered the arterial spasm disappears.

As I have already indicated, the arterial spasm may be determined by direct irritation of the stomach. In two of my cases the spasm and its accompanying paroxysm of angina were caused by palpation of the epigastrium. This illustrates the well-known nerve connection between surface and viscera—gastric disorders frequently causing surface hyperæsthesia, while in these two cases the surface stimulus produced the same effect as if a stimulus had been applied directly to that viscus.

The stimulus is also provided by the substances taken into or absorbed from the alimentary tract. These substances exercise a supreme influence in producing and in maintaining the hypersensitiveness of the vasomotor reflex, and therefore of the vasomotor centre.

This *hypersensitiveness of the vasomotor centre* will explain what has long been recognised—that paroxysms of angina have as their main determining cause physical effort, mental emotion, or digestive disturbances.

The Unifying Principle.

By the application of the facts I have brought under review the group of symptoms known as angina pectoris can be satisfactorily explained, no matter how diverse the conditions may be in which it occurs. It explains the occurrence of the symptoms without anatomical change, while at the same time it shows the importance of the presence of anatomical changes in the coronary arteries, or in the myocardium. In this connection it must always be borne in mind that even calcareous coronary arteries are not calcareous to their finer subdivisions, so that in this condition, as well as in the varying degrees of arterio-sclerosis, the power of spasm contraction is not lost—may indeed be intensified. It gives to the nervous system its due place in the production of the phenomena.

To illustrate the production of a paroxysm by mental emotion or physical effort, let me refer to Case III., in which slight emotional disturbance or slight exertion had the effect of inducing a paroxysm. The connection between the emotional centres and the vasomotor centre is a normal one, and constantly active. In this case a normal emotional impulse acted upon the hypersensitive vasomotor centre, resulting in the

arterio-cardiac phenomena of angina. This was no abnormal excitation of an emotional centre. It was a normal excitation passing on to an abnormally sensitive vasomotor centre. The vasomotor centre in this same patient was equally influenced by slight physical effort; it was not the voluntary muscle centres, nor the nerve endings in the muscles, which were unduly sensitive, but the vasomotor centre to the normal influences resulting from voluntary muscular effort.

By the cases reported I have also shown that this hypersensitiveness of the vasomotor centre, even in grave angina pectoris, can be reduced, controlled, or even removed by dietetic measures, with the result that the anginous seizures are removed or greatly modified. In cases where the arterial spasm is associated with great anatomical change, either in the myocardium or in the coronary vessels, absolute cure can hardly be looked for; but in all cases the symptoms of angina pectoris may be much ameliorated by accepting, as a working hypothesis, the propositions I have ventured to submit, and conducting the treatment in accordance with what they indicate.

There was need of a principle which, when applied, would explain the phenomena in each of the various groups into which the disorder has been divided. There was abundant clinical experience showing the remedial effect on the paroxysm of vaso-dilators; but the only explanation of the arterial spasm which up to the present time has been offered has been, to use Sir R. Douglas Powell's words, that of "a pure neurosis of the cardio-vascular system."

Owing to the varying degree of intensity of the symptoms, I would suggest that in classifying the cases the simplest distinction might be found in the terms "angina pectoris major" and "angina pectoris minor," the former being confined to those cases in which there was believed to be permanent anatomical change in the heart or its vessels.

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Meeting III.—December 20th, 1905

Dr J. O. AFFLECK, *President, in the Chair*

This was a Clinical Meeting, held in the Royal Infirmary.

I. EXHIBITION OF PATIENTS

1. *Dr Norman Walker* exhibited—(*a*) A case of MYCOSIS FUNGOIDES after treatment by X-rays, and a wax cast of the condition before treatment. He mentioned that each affected area had to be individually treated. In this case fresh lesions had appeared on the shoulder, and would require treatment. (*b*) Cases of RINGWORM which had been treated by X-rays. He discussed the advantages and disadvantages of the method. In his opinion the latter were too lightly regarded.

2. *Dr W. Allan Jamieson* exhibited a case of ADENOMA

SEBACEUM complicated with MOLLUSCUM FIBROSUM, var. PENDULUM.

C. B., 26, farm labourer, Berwickshire. Admitted to Ward 22, Royal Infirmary, on 16th December 1905. He is a well-built, rather dull, but not unintelligent man. Has suffered from epileptic fits for some years, subsequent to an injury to his head caused by a horse rearing and striking him with its foot. His mother declares that the growths on his face were not present at birth or in childhood, but only commenced about ten years ago. Some, she says, have disappeared from the sides of the cheeks, but others have come. These are now limited to the central parts of the face, the nose and adjacent parts of the cheeks. They consist of small solid tumours, from the size of a pinhead to half a pea. They are sessile, pale, or pinkish ; a few have fine arborescent vessels running over them. Under the right eye they have become aggregated into a solid mass, the size and shape of half an almond. They cause no subjective sensations, and correspond rather to Pringle's than to Balzer's type. Besides these, he has innumerable mollusca pendula on his shoulders, back, and chest, but ending about half-way down. These are a more recent development than the adenomata. They vary from minute, hardly perceptible, white points, to flat, pedunculated pouches, half an inch long. There are no areas of discolouration.

3. *Dr Logan Turner* and *Mr Alexis Thomson* exhibited a patient, male, 56 years, upon whom the operation for COMPLETE REMOVAL OF THE LARYNX had been performed. The patient had been troubled with hoarseness for three months. The left side of the larynx was found to be completely fixed ; there was infiltration of the left false cord, left arytenoid region, and the interarytenoid fold, and it was deemed advisable to remove the whole larynx. The operation was performed in December 1904. A year later he enjoyed good health, having gained three stones in weight since the operation. There was no local recurrence of the disease.

4. *Professor Sir Thomas R. Fraser* exhibited cases representative of several forms of disease. The two first are examples of peripheral neuritis, originated, in one of the cases, undoubt-

edly from arsenic administration, and, in the other case, probably from the excessive use of tobacco.

(*a*) PERIPHERAL NEURITIS CAUSED BY ARSENIC.

Jane Meade, age 14, was admitted into Ward 25, suffering from a second attack of chorea. She is a well-developed and well-nourished girl. The attack for which she was admitted on this occasion, on 17th April 1905, began about a fortnight before her admission. It then involved the left side chiefly, but before her admission the right side also became involved; and, on entering the hospital, the patient presented the usual symptoms of a moderately acute and general chorea. We found that all the sensibilities were healthy and sharp, that the heart's sounds were normal, and that all the other organs were healthy. It is noteworthy that on many occasions choreic contractions of the diaphragm and also of the heart were observed.

On the day of her admission, arsenical treatment was begun. The choreic symptoms gradually lessened, until on the 17th of May they had altogether ceased, and the patient was allowed to sit up for a short time daily. On the 25th of May, however, slight choreic movements recurred, but after five-minim doses of liquor arsenicalis had been administered thrice daily for four days, they disappeared. The patient was allowed again to leave her bed on the 5th of June, and now it was observed that a new group of symptoms had developed. Standing and walking were performed with difficulty, and the patient required to be supported when doing them. On the 10th of June a detailed examination was made. There was slight brown pigmentation of the skin of the abdomen and of the lower eyelids, and the skin of the lower and upper extremities was very dry and scaly. No paræsthesia was existent, but tactile sensibility was lost in both lower extremities below the tubercle of the tibia, and was much delayed on the thighs, hands, and forearms, while thermal sensibility was much delayed in the legs below the knees and in the feet. The muscle sense, the special senses, and the organic reflex appeared unaffected. No knee jerk could be elicited; the plantar reflex was absent in both lower extremities, but the other superficial reflexes were unimpaired. Voluntary movements were found to be much weakened. The patient could walk only when supported on each side. When she raised the legs, the feet

hung flaccidly in an extended position; when she walked, the legs were lifted high, and the toes dragged on the floor; and when she stood, the knees became bent, and she could not herself maintain a standing posture. She could not extend the toes of either foot, or dorsiflex the ankles, or evert the feet, and most of the other movements of the lower extremities were very feebly performed. Extension of the fingers and wrist was imperfectly accomplished, and there was partial drop-wrist. The muscles everywhere were flabby, and acute pain was experienced when those of the calves and forearms were pressed. It was also found, in the case of many of the muscles of the extremities, that Faradic currents elicited no contraction, while a weaker galvanic current caused contraction with anodal than with cathodal closing stimulations.

The patient, therefore, exhibited all the characteristic symptoms of multiple peripheral neuritis. As you will observe, the majority of the symptoms I have described are still present, but in a considerably lessened degree. The treatment of the peripheral neuritis—with warm embrocations, cod-liver oil, and iodide of potassium, and latterly with massage and strychnine—has, as is usual in this disease, only very slowly assuaged the severity of it. Recently a distinct improvement has become manifest, and I have no doubt that complete recovery will ultimately be produced.

It is interesting to add that between the 17th of April and the 7th of June 1905 the patient received altogether 1068 minims of liquor arsenicalis (about 2 ounces and 2 drachms), no doubt a large quantity, but large very much because of the singular obstinacy to the therapeutic influence of arsenic which this case manifested. Even after recovery from chorea had apparently occurred, and when the appearance of peripheral neuritis rendered it necessary to stop the further administration of arsenic, slight choreic symptoms reappeared on two occasions, but their duration was only brief, although bromide of potassium and phenazonum were required to remove them.

(*b*) PERIPHERAL NEURITIS AND TOBACCO.

The next case I bring forward with some reservation as to the cause. The patient whom I place before you is a married man, a hairdresser, 25 years of age. He came into Ward 26 on the 31st of October 1905, complaining of severe

pains in the legs and back and of considerable loss of power in the feet. We learned that twenty days previously, while taking a holiday at Dalkeith, he suddenly saw everything double, and this double vision made him feel giddy. He had no paræsthesia and no difficulty in walking ; and no other symptom occurred until two days subsequently, when he felt "sleepiness" in both hands, with inability to hold things without allowing them to drop, and the hands were sometimes tremulous, and difficulty was experienced in walking. In two weeks the diplopia disappeared as suddenly as it had first appeared. The other symptoms, both sensory and motor, became increased in degree ; he slept badly because of headache and racking pains in the calves of the legs, and had become quite unfit for work before entering the Infirmary. On his admission we found that the only subjective symptom was the dull aching pain in both calves. Tactile sensibility seemed unimpaired, but thermal sensibility was much delayed and often mistaken on the dorsum and toes of the left foot. The muscle sense, the organic reflexes, and each of the special senses were healthy. No skin reflex could be elicited, and the knee-jerk was absent. The patient was unable to stand or walk without support, and when supported both feet dragged behind. When lying in bed he was unable to raise himself or turn from one side to the other without the assistance of his arms. Voluntary movements were elsewhere much impaired. In the shoulder and elbow joints they were feeble, and they were slowly effected as well as feeble in the wrists, fingers, and thumbs. Similar defects were present in the hips, knees, ankles, and toes of the lower extremities. In many parts of the body, also, fibrillary twitches of the muscles were observed. The muscles themselves were soft and flabby, and when those of the thighs, calves, and arms and forearms were held firmly, severe pain was produced. It was found that all the muscles responded well to both Faradic and galvanic stimulation, and that with the latter the cathodal closing current produced contraction with a weaker current than the anodal.

Nothing worthy of note was found in the other systems. Since his admission no new symptoms have developed, but the paralysis has everywhere increased, and it will be observed also that the muscles, now disabled for several months, have become smaller and in several localities distinctly atrophied. Indeed,

the last change is now suggestive of a beginning of, or complication with, anterior cornual degeneration.

Our inquiries into the history of the patient have revealed nothing of significance in his family history, and only one circumstance of possible importance in his personal history. He has a good reputation as a steady, frugal, and abstemious man. He denies having suffered from venereal disease. His wife, however, had an abortion at the third month of her first pregnancy, ascribed by her to a strain when window cleaning, but she is now in the sixth month of her second pregnancy. No evidence of syphilis can be discovered in the patient. The noteworthy circumstance in his personal history is an exceptionally long-continued and excessive consumption of tobacco. He began to smoke Irish-roll when a school-boy of only eight years of age, to the average amount of an ounce each week. He left school, at the age of eleven, to learn hairdressing, and receiving a little more money, he gradually increased the weekly consumption until it amounted to four ounces. At 17 his financial position was still further improved, and thereupon his weekly consumption was soon raised to seven ounces. When he was 20 years old he substituted cigarettes for pipes, and disposed daily of two or three packages of Ogden's guinea gold (ten cigarettes in each package), and always inhaled the smoke into his lungs. For the last two years he has resumed pipe smoking, and has used seven ounces of the strong Irish-roll weekly. Further, since the eleventh year of his age he has used about a quarter of the total weekly tobacco in chewing, and this he also did with Irish-roll, even when he smoked cigarettes. The patient has therefore consumed a most exceptionally large quantity of tobacco from his early childhood until the beginning of this illness. Whether this great abuse of tobacco be accountable for the peripheral neuritis or be merely a coincidental circumstance, it would be rash to decide, especially as I am not aware of any case of this disease having been recorded in which tobacco has been proved to be the cause. The condition of the patient is certainly not improving, and the subsequent history will be an interesting study.

(*c*) THREE CASES OF MORBUS CÆRULEUS—

In the experience of this hospital it is an unusual circumstance to have three cases of morbus cæruleus under observation at one time, all being in one ward (25), and all admitted within a period

of five weeks. Indeed, an examination of the Reports of the Medical Registrar for the five years 1900-1904, shows that only in the year 1902 was one case admitted, while in that year 500 cases of diseases of the circulation were treated in the wards.

The first of the three patients I am able to place before you is a young girl, Annie Hastie, 6 years of age. She is stated to have been "blue" since her birth. In infancy she had paroxysms of coughing, when the whole surface became blue. When she became old enough to walk, attempts to do so caused much "panting," and she cried and sank on her knees. When at rest, while the blueness was conspicuously present on the nose, ears, fingers, toes, and all peripheral parts, it much lessened over the rest of the body, and she could sit and breathe quietly. She is well developed and well nourished, and is a chubby, happy-looking child. The fingers and toes are moderately clubbed. The appetite is poor and the temperature is generally subnormal. There has never been any œdema, and all the systems are healthy, with the exception of the circulatory system. The pulse is rapid, often 112 in the minute, but of good quality. The heart's area is widened. The second sound is accentuated and closed in all the areas. The first sound is replaced by a loud systolic bruit, best heard over the third left interspace and cartilage close to the sternum, heard also downwards to the apex and upwards to the second left interspace, and also extended towards the neck on the right side. A loud systolic bruit is also heard on the left axilla and over the left back, especially in the interscapular region.

An examination of the blood gave the following results :—

Hæmoglobin, 110 per cent. } 85 per cent. of hæmoglobin in each red blood-corpuscle.
Red blood-corpuscles, 6,930,000. }

White blood-corpuscles, 8000.

Specific gravity, 1070.

The second patient is a young woman, Melissa Sinclair, 21 years of age, who came to Ward 25 from Cumberland, on the 9th of November 1905.

When a fortnight old, she lost a large quantity of blood by the giving way of the navel ligature. Two days afterwards, the mother, while nursing the patient, heard a rhythmic blowing or "puffing" sound, which seemed to proceed from the

heart. This increased in loudness as the child grew, until in three or four years it could be heard across a dining table; and after persisting for seven years, it gradually diminished and then disappeared. The patient has all her life been subject to severe paroxysms of breathlessness and palpitation, especially on exertion, and was, therefore, never able to run about and play with other children. The paroxysms were irregular in their severity, varying from mere dyspnœa with palpitation to general convulsions with loss of consciousness. She menstruates regularly every four weeks. Besides the circulatory system, only the alimentary is disordered, there being slight dyspepsia and moderate constipation. The pulse is about 100 per min., but the rhythm changes easily. Its characteristics are those of health. None of the large external veins are enlarged, but there is marked blueness of the face and periphery. The heart is slightly widened, a systolic thrill and loud systolic bruit exist over the whole precordia, most intense over the second left interspace near the sternum, propagated to the apex and to the neck in both sides, and heard also in the left axilla and over the back. On immediate auscultation, the bruit is also heard at about two inches from the thoracic wall. The blood composition was as follows :—

Hæmoglobin, 90 per cent.
Red blood-corpuscles, 5,390,000.
} 88 per cent. of hæmoglobin in each red blood-corpuscle.

White blood-corpuscles, 8200.

Specific gravity, 1070.

There is only very slight clubbing of the fingers. As you observe, the patient is a bright, placid-looking girl, but she is very backward in her education.

This placidity of disposition is not, however, possessed by the third of these patients, Ivy Hastie, 6 years of age. Our information of her history begins when she was six weeks old, at which time she was consigned to the care of a foster-mother, her actual mother being the wife of a soldier without fixed abode, and frequently living abroad. She has been blue in the face and over all the rest of the body at least since she was fourteen days old. When six weeks old she was very thin and small, as she now is, and, although remarkably intelligent, she has always been of a singularly peevish disposition with a violent temper. The cyanosis is greater in degree than in

either of the two other patients, and involves the interior of the mouth and the eyeballs. It is much increased when the patient cries or becomes angry, and, also, apart from these circumstances, during "fits" of an epileptic character, from which she has suffered for several years. There is much clubbing of the fingers and toes.

The transverse diameter of the heart is increased. A loud systolic bruit is heard over the whole precordia, of greatest intensity at the second left chondro-sternal articulation and in the pulmonary area, whence it is propagated for two inches upwards and to the left. The systolic bruit in the tricuspid area is of different character from the bruits in the other areas, and is associated with slight pulsation and some enlargement of the right external jugular vein. The pulse is always rapid, from 96 to 128 per min., but, notwithstanding the cardiac bruits, it is regular and possesses the characteristics of health.

The examination of the blood revealed a remarkable increase of the hæmoglobin and red cells, the results of the examination being :—

Hæmoglobin, 160 per cent. } 81 per cent. of hæmoglobin
Red blood-cells, 9,660,000. } in each red blood-corpuscle.
White blood-cells, 11,000.
Specific gravity, 1075.

As has frequently been observed in this "blue-disease," the temperature is subnormal, and on several occasions it was found to be so low as 95° F.

The other systems and organs are in a healthy condition.

It does not appear that any adequate explanation has been propounded of the blue colouration or of the excessive quantities of hæmoglobin and of red blood-corpuscles which so frequently accompany it in morbus cæruleus.

(*d*) URTICARIA A FRIGORE.—The last case I wish to present to you is illustrative of a relatively infrequently encountered variety of artificial urticaria, in which the ordinary features of dermographia are not produced by the friction or pressure of a pointed surface on the skin, but only by the application of cold. The patient, a cork-cutter, 56 years of age, was admitted into the Royal Infirmary because of enlargement of the spleen and liver with irregularly occurring pyrexias, having the characters of ague. He, however, had never been abroad, and we have never succeeded in finding the malarial parasite in his

blood, even although it has frequently been searched for in apyretic periods as well as in one pyretic period which occurred since he came into hospital.

The discovery of the effects of cold on the skin was accidentally made. One morning since his admission into hospital the patient noticed that after he had rested the bared surface of one of his arms for a few minutes upon the iron bar at the top of his bed, he felt the part itchy, and, on afterwards looking at it, he observed a red elevated mark, which remained for about two hours. He reported the circumstance to Dr Fourie, the resident physician, and a number of observations were thereupon made which confirmed the patient's statements. In the first instance, an iron dumb-bell was placed on the arm for three minutes. Itchiness was felt in one minute, and when the dumb-bell was removed a bright red and slightly elevated area was seen which occupied the part of the skin that had been in contact with the iron. This area became gradually more elevated during the next few minutes. The itchiness disappeared in a few minutes, but the elevation remained for nearly two hours. A large number of other observations were made during the next few weeks. Pieces of iron, copper, silver, and gold, of various shapes and sizes, were cooled in ice and applied to many parts of the surface of the body ; without describing the results in detail, it is sufficient to say that they all succeeded in producing itchiness and similar elevations of the skin, always exactly representing the shape of the body which had been placed on the skin. The appearances are well represented in the examples that I now show you, which have been produced by these means on the arms, thorax, forehead, and scalp, shortly before the patient was brought into this theatre. I have found that they can be produced in all parts of the body except on the soles of the feet and the palms of the hands, but not readily on the hips or back. This urticaria seems to be most easily produced in cold weather. Cold, however applied, produces it, as, for example, pieces of ice or cold water in a test-tube ; but only cold objects seem capable of doing so. Heat to the greatest degree that can be tolerated altogether fails, and so do interrupted and continuous electric currents, X-rays, radium, prolonged pressure, and friction.

It may be added that the patient has recently told us that, during the last six or seven years, he had noticed that when he

washed in cold water in winter the skin of his hands and face became itchy and red, and continued so for a short time, and that the discomfort thus produced had led him to avoid, as much as possible, the use of cold water when washing in winter.

What explanation can be advanced of this effect of cold applications? It cannot be the mere result of irritation, otherwise it would also follow the application of the several forms of irritation that have been proved ineffectual to cause it. This is not a suitable occasion to discuss questions of etiology, but I would so far transgress as to express the opinion that in the blood or other tissues of this patient there is present a substance which acquires under the influence of cold the power of producing urticaria, just as there is in the blood of patients liable to paroxysmal hæmoglobinuria a substance which acquires hæmolytic properties only after it has been exposed, whether in the body or *in vitro*, to the influence of a low temperature.

5. *Mr C. W. Cathcart* exhibited a case of ABSCESS IN LIVER FROM GALL STONES.

Mrs R., aged 34, admitted to Ward 17, Royal Infirmary, on October 27th, 1905, on account of what she described as "steady tapping pain underneath the liver." The seat of pain was in a swelling which lay a little to the right of the umbilicus. Seven years before admission she had suffered from "cramps in the stomach" associated with occasional bilious vomiting. This went on for about two years when great improvement resulted from the introduction of a uterine pessary. The gastric pain was thought to have been reflex, resulting from displaced uterus. After that she had occasional pains over the right lower ribs and in the region of the right kidney. These pains were relieved when the bowels were freely opened. About a month before admission she had been attacked by severe pain in the abdomen. This has continued in the same place and has latterly kept her awake at night.

On admission the temperature varied from 96.4° to 99°, and her pulse from 86 to 96 beats per minute. The abdomen moved freely in respiration. A swelling over the right rectus was perceptible to the right of the umbilicus. This swelling, which was very tender to the touch, moved with respiration; the lower margin was irregular. Percussion note was dull from

the region of the swelling to the lower border of the ribs. Next day the pulse rose to 112, and the area of abdominal pain increased. The abdomen was opened, and the swelling was found to be caused by a large abscess in the liver. The lower margin of the liver reached to a point below the level of the umbilicus, and it was overlapped by the colon. The abscess had begun to leak into the abdominal cavity near the empty and contracted gall-bladder.

The region of the abscess was packed round with gauze swabs, and the abscess was then incised, and found to contain six facetted gall stones, besides several ounces of thick pus. A drainage tube was put in, in the pouch below the liver, and brought through an opening made in the loin. Another drainage tube was left in the abscess cavity, and brought out at the anterior wound. It was packed round with gauze. The upper and lower ends of the abdominal incision were then closed.

The patient has made a slow, though good, recovery.

As illustrating the value of the posterior drainage opening, about ten days after the operation the posterior drainage tube was withdrawn. The temperature, which had remained normal till then, rose to 100.6 next morning and 100.8 in the evening. On re-inserting the tube the temperature at once fell. Now the posterior wound is healed, and the anterior wound very nearly so.

The diagnosis in this case was not made out till the abdomen was opened. The position of the liver was unusual, and the absence of symptoms pointing to the ulceration of the gall-bladder and escape of the gall stones was remarkable, unless this had taken place during the illness, which had begun four weeks before admission to the Infirmary. The rise of pulse which had determined the operation seemed to have been due to the escape of pus into the general peritoneal cavity. Otherwise the pulse and temperature had not indicated suppuration.

6. *Mr Alexis Thomson* exhibited—

(*a*) A man, aged 50, who had suffered from PRURITUS ANI for eighteen years, and who had tried every known remedy without relief. Ball's operation was performed in June, since when the pruritus had entirely disappeared.

(*b*) A man, aged 35, nine months after REMOVAL OF THE

CÆCUM and associated lymphatic glands for columnar-celled carcinoma. The ileum and ascending colon were joined end-to-end by suture. The patient had gained two stones in weight since the operation.

(*c*) A man, aged 31, five months after RESECTION OF THE PYLORIC SEGMENT OF THE STOMACH FOR CANCER. He had suffered from gastric symptoms for fourteen months, had recently lost weight, and shown signs of pyloric obstruction. The stomach-wash showed absence of free HCl, and presence of lactic and butyric acids. On opening the abdomen the cancer was found to be limited to the pyloric segment, but there were indurated glands between it and the pancreas. Billroth's second operation was performed. He has gained two stones in weight, and never suffers from indigestion.

7. *Mr Scot Skirving* exhibited—

(*a*) A PATIENT AFTER PROSTATECTOMY BY FREYER'S METHOD. The case was shown as an example of the very favourable after-result that follows this operation in many instances. The patient, a man of 59 years, had suffered from marked prostatic symptoms, with much pain. Operation was performed on October 20th, and occupied seventeen minutes. The wound was completely healed on the twenty-seventh day, and the patient had resumed work as a dock labourer (lifting heavy sacks, etc.) on the thirty-fourth day. No residual urine was now present, and the only symptom complained of was slight frequency of micturition. Mr Skirving had found attention to two points of value during enucleation of prostates: first, to insert one's own finger in the rectum—*i.e.* not to employ that of an assistant; and secondly, to have the patient placed as low as possible.

(*b*) A MAN, AGED 52, WITH DISLOCATION OF BOTH ENDS OF RIGHT CLAVICLE. The case was shown on account of its rarity. The cause of the accident was a fall backwards from a pair of steps eight feet high. When first seen, the clavicle could be pushed back and forward between the examining hands. In other respects the lesion resembled forward dislocation at the sterno-clavicular joint, there being almost no displacement at the acromial end, and the deformity still present was at the sternal extremity. Very little disability had resulted.

Treatment had been three weeks' confinement in bed with a pillow between the shoulders.

(*c*) A BOY AFTER OPERATION FOR ACUTE OBSTRUCTION DUE TO TUBERCULAR STRICTURE, WITH SUBSEQUENT ILEO-COLIC ANASTOMOSIS. When operated on, ten months ago, fœcal vomiting was present, with marked intestinal peristalsis. Per rectum, a mass like a boy's football impinged against the finger with each spasm. This turned out to be a loop of enormously distended ileum immediately above the stricture, which was some two feet from the cæcum, and which just admitted a pencil. The distended loop was about the circumference of a distended transverse colon in an adult. (Age of patient, 13 years.) The stricture was excised, and both ends of the divided gut brought out of the wound. Ten days later the proximal end was implanted by lateral anastomosis into the ascending colon, the distal portion being excised close to the cæcum. The cæcum was also the seat of tubercular disease, but was not interfered with. As in many of such cases, it was found impossible to bring the edges of the abdominal wall together. The wound was accordingly merely packed with gauze, and subsequently strapping applied. It was interesting, therefore, to note that no hernia had occurred. The usual rapid increase in weight had taken place, and the boy's condition was now all that could be desired.

8. *Dr Alexander James* exhibited—(*a*) CASE OF EPIDEMIC CEREBRO-SPINAL MENINGITIS. (*b*) CASE OF SPRUE. (*c*) CASE OF MALTA FEVER. (*d*) CASE OF PNEUMONIA INTERCURRENT IN DIABETES MELLITUS.

9. *Mr F. M. Caird* exhibited—

(*a*) A CASE OF COXA VARA.

T. H., a healthy lad, aged 17, began to limp, without obvious reason, six months ago. Prior to that he was perfectly well, and had sustained no injury. He now has marked shortening of the right leg, about one inch ; eversion and adduction. He cannot flex the hip-joint. There is no history of pain. The skiagram reveals characteristic curvature and descent of the femoral neck.

(*b*) NEPHRECTOMY FOR RENAL CALCULUS.

Mr J. E., æt. 35, complained of lassitude, thirst, and the

presence of blood in the urine. His symptoms began fifteen years ago, when blood was first seen in the urine. It did not recur for seven years, when it was then associated with frequency of micturition, and he was treated for Bright's disease. The frequency of micturition continued. Last year he found that he could provoke the bleeding by severe muscular exercise. There was no renal tenderness or swelling. Luys' separator showed that the function of the left kidney was defective, and that blood came from it. Lumbar incision revealed a large branching calculus, which filled the pelvis and calyces, and necessitated nephrectomy.

(*c*) PATIENT AFTER OPERATION FOR CARCINOMA OF THE RECTUM.

Mr T. M., æt. 59, had a large high-placed carcinoma recti removed in December 1901 by the method of Kraske. It was impossible to unite the ends of the divided gut, and he accordingly left the hospital with the sphincteric segment *in situ*, but functionless, and an artificial anus above that in the sacral region. In 1904 the disease returned, and a large fungating mass required the entire removal of the sphincteric segment in October 1905. The patient is now well and strong, and the artificial anus gives rise to but little inconvenience.

(*d*) COLOSTOMY FOR INOPERABLE CARCINOMA RECTI.

Mr T. W., æt. 58, suffering from extensive inoperable carcinoma of a year's duration. Colostomy in the left linea semilunaris, October 1902. Complete division of the sigmoid colon, and formation of an artificial anus, which gives the patient no trouble, unless after an indiscretion in diet.

(*e*) LARGE RETRO-PERITONEAL LIPOMA.

Mr D., æt. 29, suffered from a huge abdominal tumour of a year's duration. It was associated with a sense of constriction and tightness, with pain after meals, and occasional coffee-ground vomiting. On operating, a huge retro-peritoneal lipoma was found, apparently springing from fat in the neighbourhood of the left iliac vessels. It weighed over 17 lbs., and displaced the stomach, intestines, and bladder, and had formed numerous adhesions. The patient made a good recovery.

10. *Mr George Chiene* exhibited—

(*a*) A child, five years of age, after TRANSPLANTATION OF TENDONS for deformity due to infantile paralysis. When first

seen by him the foot was markedly everted, the toes pointed, and, when the child walked, the anterior portion of the foot dragged on the ground. On examination, all the muscles below the knee, except the peronei, were wasted, and the electrical reactions were lost. The operation consisted first in dividing the peroneus longus and brevis close to the insertion of the brevis, then an incision was made above and behind the external malleolus, and the tendons drawn out through the upper wound. The tibialis anticus and posticus were exposed on the inner aspect of the foot. A channel was then made over the dorsum of the foot, and the peroneus brevis tendon passed along it and stitched to the tendon of the tibialis anticus. In the same way the peroneus longus was passed between the tibia and the tendo achillis and stitched to the tendon of the tibialis posticus. Mr Chiene mentioned that the greater part of the operation had been performed under spinal anæsthesia, but, owing to the age of the child, he had given too small a dose to allow of its completion. Although the foot was still slightly everted, and the patient walked with a limp, she was able to completely lift the foot from the ground.

(*b*) A young woman, aged 21, on whom he had operated for CLEFT PALATE a few months previously. He showed the case as the condition in a patient of that age, in which operation was advisable, was unusual. The palate had completely united, and the patient's articulation had already improved. The only difficulty at the operation was somewhat troublesome hæmorrhage.

(*c*) A young man, on whom he had operated seven months previously for PERFORATED GASTRIC ULCER and GENERAL PERITONITIS. For over two years he had been troubled with indigestion and constipation. Forty-eight hours previous to admission perforation had occurred, and when seen the patient was suffering from general peritonitis. When the abdomen was opened large quanties of pus containing flakes of lymph escaped. An ulcer was found on the anterior aspect of the stomach near the cardiac orifice. Owing to the great friability of the stomach wall in the immediate vicinity of the ulcer difficulty was experienced in invaginating it. A second opening was made above the pubis and the abdominal cavity and pelvis thoroughly irrigated. The upper wound continued to discharge for some

weeks, but finally closed and gave rise to a broad flat, though firm, cicatrice.

11. *Mr J. M. Cotterill* exhibited—

(*a*) Cases of OPERATION FOR EXOPHTHALMIC GOITRE. In both these cases the patients were women aged about 30. The right lobe had been excised under local anæsthesia, and the patients had made a good recovery.

In both instances the disease was very pronounced, the pulse rate before operation being from 160 to 175 per minute. In both instances considerable improvement had resulted, and it was hoped that further benefit would ensue.

(*b*) A case of MALIGNANT DISEASE OF THE ŒSOPHAGUS, in which the patient was losing weight at the rate of $2\frac{1}{2}$ lb. a week. Intubation had been performed, and the patient was doing extremely well, having put on $2\frac{3}{4}$ lb. in the first six days after intubation.

(*c*) A case of MARKED HYPERTROPHY OF HANDS AND FEET in a boy aged 18. It was pointed out that the case was not one of "acromegaly" nor of "pulmonary hypertrophic osteo-arthropathy." The exact etiology and pathology of the case was uncertain. The venous system was widely varicose, and a considerable part of the upper and lower extremities was affected with superficial nævus.

(This case was shown by Mr Cotterill for Dr G. A. Gibson, who was unavoidably absent.)

Meeting IV.—January 17th, 1906

Dr J. O. AFFLECK, *President, in the Chair*

I. ELECTION OF MEMBERS

Henry John Dunbar, M.D., 1 Kew Terrace; D. M. Chrichton, M.B., Burgh House, Maryhill, Glasgow; William H. Myres, M.D., Fort Wayne, Indiana, U.S.A.; and Herbert Sloman, L.R.C.S.L., M.R.C.S., 9 Murrayfield Avenue, were elected Ordinary Members of the Society.

II. Exhibition of Specimens

1. *Dr James Cameron* exhibited a specimen of STRANGULATED RIGHT TESTICLE from case of STRANGULATED HERNIA AND TESTICLE, in an infant of six weeks.

In this case when the child was seen the day previous to operation there were indefinite symptoms of uneasiness with depressed fontanelle.

Next day the supervention of apparently fæcal vomiting led to the discovery of strangulated hernia and presumably strangulated testicle, the scrotum showing a red blush.

On cutting down, etc., the bowel was found to be very tightly strangulated and covered with lymph, while the testicle and veins of the cord were quite black.

The sac was isolated, cut away from its congenital attachment to the testicle, and the proximal end, after being twisted up, placed under cover of the transversalis, the canal being closed by the usual method as in radical cure. The cord was previously cut short at the internal ring. Dilute suprarenal extract having been injected previous to making the incision, there was no trouble in the way of ligaturing or twisting bleeding vessels. Recovery was uneventful, and result excellent.

2. *Dr Douglas Wilson*, Harrogate, exhibited RADIOGRAMS OF HANDS from a case of tophaceous gout.

II. Demonstration

Capt. E. D. W. Greig, I.M.S., gave a lantern demonstration of "SLEEPING SICKNESS IN UGANDA."

III. Original Communications

1. THE USE OF STOVAÏNE AS A SPINAL AND LOCAL ANÆSTHETIC

By GEORGE CHIENE, M.B., F.R.C.S.Ed., Assistant Surgeon, Royal Infirmary, Edinburgh

MR PRESIDENT AND GENTLEMEN,—The use of a General Anæsthetic in surgical procedures, no matter what general anæsthetic is used, is always a source of possible danger to the patient; and, now that the dangers due to, or in consequence of, the actual operative procedure have, in the majority of

cases, been reduced to a minimum, the anæsthetic may be regarded as the most dangerous factor in many operations.

Not only have we to consider the immediate dangers of the anæsthetic, but also the remote dangers, which, until recent years, have been insufficiently recognised.

At intervals, various local anæsthetics have been introduced, and great advances have been made in the methods of producing local anæsthesia, both regionally and by infiltration ; but these methods have their limitations.

Bier first attempted the production of anæsthesia by spinal injection.

Cocaine was used, but, as the after results were often of a most unpleasant, not to say dangerous nature, the method for a time fell into disuse.

On the introduction of adrenalin, further attempts at spinal anæsthesia were made, the adrenalin being added to cocaine or some other local anæsthetic.

This method has received still further attention since the introduction of Stovaïne.

In February 1904, Fourneau (1), a French chemist, first publicly brought before the profession a series of chemical substances, among them the hydrochlorate of amyleine, which he named Stovaïne.

For some time previous to this date its action had been tested on man. Reclus (2) tried it as a local anæsthetic in October 1903, and shortly afterwards Chaput (3) first used it as a spinal anæsthetic.

Stovaïne is derived from tertiary amylic alcohol ; it crystallises in little brilliant flakes, resembling cocaine. According to most authorities it is a vaso-dilator, but Pouchet (4) denies this ; it is certainly not a vaso-constrictor, and has an antithermic action, points to be remembered when we compare its toxicity with cocaine. Stovaïne has also evident antiseptic properties. It is extremely soluble in water. Aqueous solutions can be boiled without interference with its action.

Solutions slowly decompose when heated to 120° C. in an autoclave, but can withstand a temperature of 115° C. for 20 minutes.

A point worth noting in regard to the chemical formula is that Stovaïne has no carbon ring ; in eucaine there is one, and

in cocaine, two. This proves that its presence is not, as has hitherto been supposed, an essential in substances having anæsthetic properties.

Stovaïne, if injected into animals in sufficient strength to cause death, gives rise to paresis of the hind legs, tonic spasms, followed by clonic spasms of a violent nature, coma, and death, in the order mentioned.

The most evident post-mortem changes are marked congestion in all the organs and dilation of the peripheral and deep vessels.

Regarding the therapeutic applications of Stovaïne, one is instinctively led to compare it with cocaine, the actions of which are already so well known.

Stovaïne slows the pulse and induces contraction of the pupil; it does not cause pallor but often flushing; there is no dyspnœa, oppression, uneasiness, or excitement. There is no tendency to syncope.

Its action, in some instances, is just as efficient as cocaine; in others, nearly so. It is most certainly much less toxic, and therefore can be given in larger doses if required.

With regard to the after results, there is usually a slight rise of temperature, which may last for some days (although, as previously mentioned, a toxic dose causes a rapid fall of the temperature). There is little tendency to sickness or vomiting; headache and spinal neuralgia are rarely present; if present, often slight. The general condition is good, and the patient can enjoy an ordinary meal. In rectal cases, retention of urine is the exception.

Regarding the early literature, Fourneau's original paper on certain amino-alcohols has already been mentioned.

Billon (5), in March 1904, gave a detailed account of the physiological properties of Stovaïne, the greater portion of which has been fully detailed by Luke (6) in the *Scot. Med. and Surg. Journ.*

Reclus (2), July 1904, gives the results of his work for the previous nine months with Stovaïne as a local anæsthetic.

He used Stovaïne in doses twice the strength that he was accustomed to use cocaine, without any bad results.

Chaput (3), in May and November 1904, gave the results of his observations, having used Stovaïne both as a local and as a spinal anæsthetic. He considers Stovaïne slightly inferior

in its anæsthetic action to cocaine. He maintains that many laparotomies can be performed under spinal anæsthesia with Stovaïne, if the patient is not emotional. For spinal anæsthesia he uses doses of 2 to 8 centigrammes. In 100 cases, 45 operations were performed on the legs, 26 on the perineum, 29 on the abdomen.

Among the abdominal operations were hydatid cysts of the liver, hernias, abdominal hysterectomies, appendectomies, pyosalpinx, and ovariotomy.

He states that all operations on the legs and perineum can be performed with spinal anæsthesia, but is of opinion that it is not good for extensive laparotomies. It can be used for appendicitis during the quiescent stage and for inguinal hernias. It is also of use in fractures, both simple and compound, and for dislocations of the lower limbs. Although the muscular tonicity remains, the reflex contractions, which are the true cause of the difficulties of reduction, are got rid of. Among other conditions, he reduced a dislocation of the hip in a young and muscular man. With doses ranging from 2 to 8 centigrammes he obtained 3 total anæsthesias including the head, 4 as far as, and including, the arms, 12 to the middle of the sternum, 13 to the xyphi-sternum, 9 to the epigastrium, 26 to the umbilicus, and 20 to the iliac spine.

He allows that the value of this method may be disputed with regard to abdominal surgery, but maintains that good results can always be obtained anywhere below the umbilicus. The height of the anæsthesia varies with the amount of the drug injected, and in all his failures only a small dose had been given. The sensitiveness of the subject must also be taken into account.

Chaput has injected 4 centigrammes for laparotomies below the umbilicus, and if, during the next 10 or 15 minutes, the anæsthesia appeared insufficient, a further dose was given. This had the desired effect. Young adults need rather larger doses. In one of his cases the pulse fell as low as 40 beats per minute, in 4 of his cases nausea was observed, in 4 vomiting, and in 10 profuse perspiration, in 7 pallor of the face, and in 5 mental distress. In his experience, the after results of Stovaïne are much less severe than those of cocaine. In his 100 cases, headache was observed 30 times on day of operation, 6 times on next day, 5 times on the third day, and once

on the fourth. It was always relieved by the application of ice. Neuralgia was more frequently met with. On the day of the operation 51 times, on the next day 29 times, on the third day 3 times, on the fourth twice, and on the fifth once.

Vomiting was noted 4 times on the first day, 6 times on the second, and once on the third. Nausea 7 times on the first day, 4 times on the second, and twice on the third.

In the majority of cases the temperature rose to about 100° F., but fell in a few hours. After cocaine the temperature is often high the following day. He states that Stovaïne has such great advantages over cocaine that it should always be used in preference. He uses Tannon's modification of Tuffier's needle.

He describes the case of a patient 66 years of age, in which 7 centigrammes of Stovaïne, taken from a "defective old stock," were injected. The pulse stopped, and all the reflexes were lost. Caffeine, ether, artificial respiration, etc., were resorted to. The pulse returned. The same afternoon the patient was able to speak, but suffered from a low delirium which continued for some days, at the end of which time he completely recovered.

He does not care to employ Stovaïne in patients over 65 years of age, but if used in old people it should be given in small doses. He describes another case, a woman 52 years of age suffering from arthritis of the hip and great pain in the legs. Only 1 centigramme was injected. No anæsthesia was obtained, but two hours later bilious vomiting occurred. The temperature rose to 104° and the patient became somnolent. The next day the temperature remained high, the somnolence increased, and she died at one o'clock. He considers this case to be of an entirely different nature to the previous one, which he looks upon as a true case of intoxication.

I have quoted Chaput's paper somewhat fully, as it records the first results obtained with Stovaïne as a spinal anæsthetic.

Lapersonne, about the same time, first used it as an anæsthetic in ocular surgery. It has also been used in dentistry, midwifery, and for neuralgia.

Sonnenburg (7) has given us a detailed account of his work with Stovaïne as a spinal anæsthetic, the results being similar to those of Chaput. He regards the use of Stovaïne

for spinal anæsthesia as an important advance in the whole question of narcosis, and maintains that not only operations on the extremities, but also abdominal operations as high as the costal arch, can be performed painlessly.

In 56 cases he had 11 failures, and in 1 case death resulted. This he reports fully. The patient suffered at the time from septic peritonitis. Ten days after the operation the temperature and pulse rose. Two days later the patient developed twitchings of the hands and arms and stiffness of the neck. On the thirteenth day after the operation the patient died. At the post-mortem examination localised abscesses were found in the abdomen. The inner surface of the dura was covered with pus ; the posterior cranial fossa was also affected.

He considers the lowest age limit to be about 14 years, and the upper age limit to be about 60. He goes somewhat fully into the various theories regarding the innervation of the peritoneal cavity.

In conclusion he gives a summary of his cases.

Tuffier (8), in June of 1904, describes his old technique, substituting Stovaïne for cocaine. He considers its anæsthetic properties both as regards intensity and duration as good as cocaine. Further, that if no fault be made in technique, the anæsthesia is certain.

He is of opinion that no bad results follow from spinal anæsthesia, having anæsthetised over 2000 cases by this method, and having watched many of his cases for over four years. With Stovaïne he had then operated on 89 cases, including operations on the uterus and the appendix. He regards the introduction of Stovaïne as an important advance in spinal anæsthesia.

Silbermark (9), writing on spinal analgesia, does not consider old age as a contra-indication. Bier (10) describes a record of 102 cases. He, too, considers this method a good one for old and weak patients. He considers it contra-indicated in laparotomies, but useful in rectal cases. In 7 of his cases vomiting occurred, in 1 slight collapse, in 10 unpleasant after effects occurred. Columbani (11) maintains that debility and old age are not contra-indications for spinal anæsthesia, but that a marked degree of arterio-sclerosis is. It is contra-indicated in children and in persons suffering from nervous

diseases and syphilis. Reclus (12) is not very enthusiastic regarding spinal anæsthesia, and considers that many operations performed by this method might be done with local anæsthesia. Filliatre (13), in May 1905, read a paper on spinal anæsthesia induced by cocaine, and in it mentions that Stovaïne was used in some cases, but was unsatisfactory both at the time and afterwards. Time does not permit of any mention of other foreign literature on the subject, and I can find no original articles on Stovaïne as a spinal anæsthetic in this country.

In October 1904 I heard of this new anæsthetic, and being interested in local anæsthesia I obtained some. Since then, on various occasions I have used it combined with hemesine as a local anæsthetic when occasion offered.

In the spring of 1905, when in Paris, I saw M. Tuffier operate on several cases in which Stovaïne was used as a spinal anæsthetic, and at that time obtained the solution and instruments as used by him.

My spinal cases are few in number, chiefly owing to the fact that with a new drug I have been particular to obtain the permission and full approval both of the patients and their doctors.

As previously mentioned, the usual amount of Stovaïne used for spinal anæsthesia ranges from 2 to 8 or 10 centigrammes. So far, I have found that I require a relatively larger dose than that usually recommended to get the anæsthesia to pass to the required height. I have used Tuffier's solution, 10 per cent. of Stovaïne in sodium chloride. This is supplied by Billion in ampuls, one of which contains half a c.c., representing 5 centigrammes of Stovaïne.

This solution is now supplied in ampuls of a more convenient shape than those which I originally obtained.

Bier has recently suggested a formula which differs from Tuffier's solution in that it contains adrenalin. He maintains that its specific gravity is nearer that of the cerebro-spinal fluid than Tuffier's, and consequently there is less chance of headache and spinal neuralgia following its use. It is also said to maintain the anæsthesia for a longer period.

I have used Tuffier's needle and an all glass syringe, which can be boiled. If boiled with other instruments, all

traces of soda must be removed both from the syringe and from the needle before using.

The patient's lumbar region is prepared the night previous to the operation in a similar fashion to the part to be operated on, and is then covered with a dry sterilised dressing. This is removed in the theatre, and the skin again thoroughly purified. I regret that lumbar puncture was not practised in the medical wards of the hospital when I was a resident as it now is. If it had been, I feel sure that the results of my cases would not have been so varied as they are. In other words, the failures were due rather to the faulty technique of the operator than to any fault of the method. Sonnenburg and Columbani both mention faulty technique as the probable cause of failures. When one is about to inject the drug the needle is first fixed to the syringe, an assistant breaks the ampul, the needle is inserted and the required amount of fluid drawn into the syringe. Remove the needle and lay the syringe carefully down on a sterilised towel. The patient bends as far forward as possible. Tuffier's line is found by ascertaining the position of the highest points of the iliac crest, and drawing an imaginary line between them. This crosses the spine of the fourth lumbar vertebra. The needle is inserted about a centimetre from the middle line at a slightly lower level, inwards and forwards for a distance varying from 5 to 7 centimetres, depending on the muscularity of the subject.

With the patient in the above position, the instruction which is sometimes given to pass the needle upwards is, I think, misleading.

In order to facilitate the introduction of the needle, I have had a detachable metal handle made, which I have found useful.

One knows at once when the needle passes through the tough ligamentum sub-flavum.

On passing the needle a little further on, the cerebro-spinal fluid should escape when the handle is removed. The syringe is then fitted to the needle, and the piston withdrawn. Some cerebro-spinal fluid is in this way mixed with the solution in the syringe. The combined contents are then slowly injected, the needle rapidly removed, and the puncture sealed. The needle may strike bone, in which case it must be partially

withdrawn and reintroduced at a different angle. It may be necessary to withdraw the needle and make a fresh puncture at a different level. Should a vein be punctured, a fresh needle should be introduced at another point. Although the needle enters the canal, no cerebro-spinal fluid may escape, in which case the needle is probably blocked and a stilette should be passed along it. Puncturing the skin in this region is not at all painful. If the injection be successful the patient will, in a few minutes, begin to feel a condition of "pins and needles" in the legs, or he may complain that his lower limbs are going to sleep. Those premonitory signs are shortly followed by an inability to lift the limb off the table, and in about seven minutes sensation to pain is lost, although sensation to touch remains.

The anæsthesia commences in the perineal region, then affects the toes and feet, and passes gradually up the limb. The height and duration of anæsthesia depend as a rule on the amount of the drug injected.

The anæsthesia disappears in the reverse order to that in which it set in, sensibility returning last in the region of the perineum.

I have attempted spinal anæsthesia with Stovaïne in 14 cases. In 2 I failed to reach the spinal canal; in a third the needle entered the spinal canal, but, owing to blocking of the needle, I was unable to make the injection. With regard to this case, it is of interest to know that some days later she developed severe spinal neuralgia and occipital headache. This cannot possibly be put down to the drug, as none was injected.

Turton (14), writing on lumbar puncture for diagnostic purposes, states that "troublesome after effects are rare, though headache, nausea, vomiting, and convulsions have been produced." In 3 other cases chloroform had also to be given, in every case due to an insufficient dose. In one, a man of 23, suffering from inguinal hernia, 4 centigrammes were injected. Anæsthesia developed only as far as the thigh. A second injection was attempted, but the syringe broke, and chloroform was given. Little chloroform was required.

Another was that of a child 5 years of age, in which the dose was 1½ centigrammes. In this case transplantation of tendons was carried out, and the greater part of the operation

done under spinal anæsthesia. Before the operation was completed the anæsthesia began to pass off, but a few whiffs of chloroform sufficed to finish the operation. The third was a case of sigmoidostomy, in which the anæsthesia only ascended as far as the thigh. He also only required a little $CHCl_3$. In another case, suffering from tubercular glands in the groin which had suppurated, the anæsthesia was not complete.

In the remaining cases the anæsthesia was absolutely satisfactory. One girl, operated on for severe traumatic flat foot, complained, but could not tell what was being done. As soon as the hammer and chisel were used she stopped complaining and began to take an interest in the proceedings. Perhaps it is needless to state that the field of operation is screened from the patient's sight. The other cases included patients up to the age of 61. Two suffered from hæmorrhoids, one from severe varicose veins of both legs, one from double inguinal hernia, both sides being operated on at the same time, and one from tuberculous foci. This patient was operated on twice; on the second occasion the knee-joint was opened into and the patella sawn through. In the hæmorrhoid cases the stretching of the sphincter gave rise to no inconvenience whatever. No bad results occurred during the operations, but the pulse of the child already mentioned became rather feeble as the anæsthesia passed off. No bad after results occurred. One patient suffered from slight spinal neuralgia, and one from headache. Both yielded to treatment. I regret that my cases are so few in number and so varied in results, but I am perfectly satisfied that with a little more experience, both in technique and in the amount of drug required, failures will be exceptional. I hope at a future date to submit a further series of cases.

I have avoided operating in this manner on cases in which sepsis was present. The death described by Sonnenburg appears to me to be one in which organisms, previously circulating in the blood, had settled down in the spinal cord; possibly this is accounted for by the local traumatism set up by the puncture.

It may be argued that one is putting the patient to an unnecessary risk by injecting drugs into the spinal canal, but one also runs risks when giving general anæsthetics. For those patients who dread the latter more than the operation, spinal anæsthesia is particularly useful.

Lumbar puncture is now a routine method carried out for diagnostic purposes in medical cases, and accidents are rarely met with.

At one time surgeons looked upon the peritoneal cavity as sacred ground. Now all this is changed. May it not be that lumbar injections will in the future be more freely used?

It is too soon yet to compare the risks with those of a general anæsthetic, but it must be admitted, I think, that this method may be preferable in many cases.

I have purposely detailed all the drawbacks met with, but I am convinced that for operations on the perineum and lower limbs this method has many advantages, both during and after operation, over a general anæsthetic.

In regard to Stovaïne as a local anæsthetic, I have not time to go into the literature. I have used it both with hemisine and by itself, and have obtained better results with the mixture. In one case in which I used Stovaïne alone it apparently was an absolute failure. I have not found it superior to eucaine β and hemisine, to which I have referred on a previous occasion (15). Among the cases I have operated upon by this method are the following:—

Multiple wens of scalp.

Large single wen of scalp.

Tracheotomy for cut throat and stitching up divided larynx.

Amputation of great toe at metatarso-phalangeal joint.

Excision of fatty tumour.

Excision of recurrent nodule of carcinoma.

Double vasectomy.

Removal of a portion of needle from a foot.

Removal of portions of a rib infected with tubercle, and scraping the abscess connected therewith.

Excision of a large mass of thrombosed veins.

Opening up a whitlow and removing a necrosed phalanx.

Sinclair (15) of New York, writes a paper on gangrene of the skin following the use of Stovaïne, and cites cases in which trouble had occurred. His conclusions are as follows:—

1. That it has not the lasting anæsthetic properties possessed by cocaine.

2. That it does produce intoxication when injected subcutaneously.

3. That it is a most dangerous and unreliable drug, producing chronic œdema and gangrene of the parts into which it is injected when used in the strength of a 2 per cent. solution.

4. That it interferes with and considerably delays the healing process. Luke (6) states that its one disadvantage is that it cannot be used along with adrenalin solution, for if it is, the skin at the point of injection becomes gangrenous.

I cannot agree with the above statements, and in the cases in which I have used a combination of the drugs mentioned I have not had a single case of gangrene of the skin.

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2. SPIROCHÆTA PALLIDA (SPIRONEMA PALLIDUM) IN SYPHILIS

By Theodore Shennan, M.D., Edin.; F.R.C.S., Edin.; Senior Pathologist, Royal Infirmary, Edinburgh ; Lecturer on Pathology and Bacteriology, School of Medicine of the Royal Colleges, Edinburgh, etc.

Syphilis has always been regarded as a disease of an extraordinarily infective or contagious character, hence it is not remarkable that a contagium vivum has long been suspected to play the principal rôle in its etiology. Krzysztalowicz and Siedlecki point out that in the sixteenth century Kirscherius and David Abercromby claimed a "contagium animatum" as its cause, and Deidier (1723) spoke of "animalcula numerosa," these terms no doubt being used without much appreciation of what they imply in more modern times.

The list of causes assigned of late years is a formidable one : bacteria of all kinds, cocci, bacilli including the ubiquitous pseudo-diphtheria bacillus, and Lustgarten's bacillus, which for a time had an unsettled reign as the "syphilis bacillus," but which is now recognised to be probably identical with the smegma bacillus. Lassar states that more than twenty-five causes of syphilis have been described during the last three decades. Niessen in 1899 described a bacillus not stained by Ziehl's method but Gram-positive. This inoculated into rabbits produced ulceration, swelling of lymphatic glands, and lesions of the central nervous system in which a prominent histological feature was a peri-arteritis analogous to that found in syphilis in man. Kuznitzky described comma-shaped bodies and bodies like spirilla. Lostörfer described small refractile bodies in the blood of syphilitics which became less numerous during mercurial treatment. Winckler found peculiar spherical bodies in syphilitic local lesions and in glands.

Several authors have believed that the causal agent is protozoal—*e.g.*, Hügel, Horand with his "hémoprotiste," Holzhäuser ; lately Schüller and Stassano, the last named figuring protozoa in all stages of development. Still more recently Siegel has described the minute cytorrhyctes luis, a flagellated protozoon, and he claims that it occurs constantly in syphilitic lesions. This has not been found by more than one or two independent observers, and Metchnikoff says that Siegel's descrip-

tions and figures are sufficient to prove to him that this body is of no importance if it is not an artefact.

Neisser also discusses this question. He states that Siegel's bodies can be found in normal blood, and emphasises the fact that he claims to have produced syphilitic lesions in rabbits, which are generally considered to be immune from syphilis. To prove the point Neisser inoculated several rabbits with syphilitic material, and then after several weeks inoculated monkeys with portions of their kidneys. No lesion resulted. His experiments are strongly suggestive, but not absolutely convincing. I have seen peculiar rounded bodies in films from indolent buboes, like those described by Winckler, and which are possibly degenerated cells. They usually stain bluish with Giemsa's solution. More interesting than these are loop-shaped bodies, staining a bright pink with Giemsa's solution, and found in the same situations (see case 17). The opinion has been expressed by several, including Lassar, that some of these bodies—*e.g.*, Siegel's—may turn out eventually to be related developmentally to the spirochætæ of Schaudinn and Hoffmann. Krzysztalowicz and Siedlecki have seen similar loop-shaped bodies in glands.

The Imperial Board of Health in Berlin commissioned Schaudinn, a biologist already well known by his researches on the malarial parasite, trypanosomes, etc., to investigate Siegel's results, and he requested Hoffmann, a skilled syphilologist, to assist in the research. In a very short time they announced the discovery of spirochætæ in primary and secondary syphilitic lesions. It is a curious fact that Siegel and his cytorrhyctes are not, so far as I can find, referred to at all in the papers they have published.

The possibility of spiral forms having a causal relationship to syphilis was first hinted at by Donné in 1837. He found and figured forms to which he gave the name "vibrio lineola." Neumann refers to this observation in his text-book on syphilis (1899). Possibly the vibrio lineola may correspond to what is now termed "spirochæta refringens."

Metchnikoff and Roux in 1902 discussed the possibility of spirilla smaller than those of Obermeier or those discovered in a disease of fowls in Rio de Janeiro being the cause of syphilis, and came to the conclusion that the agent was more probably a non-motile microbe. Since Schaudinn and Hoffmann

announced their discovery Bordet and Gengout state that in 1902 they had seen these same spirochætæ in a case of syphilis, but as they failed to demonstrate them in other cases they concluded that they were of no practical importance.

Gathering together the details from a not quite complete survey of the literature[1] on the subject, and from my own experience, I may now piece together an account of the characters of these spirochætæ as at present understood. The names "spirochæta pallida," or "spironema pallidum," and "spirochæta refringens" are descriptive, the former staining faintly, the latter being markedly refractile in the living state. The *spirochæta pallida* is an extremely delicate organism, weakly refractile when living, vigorously motile, stained with considerable difficulty and difficult to demonstrate, requiring a magnification of at least 800, with good illumination. It is long, extremely thin and filamentous, of a distinct spiral or corkscrew shape, both when living and when fixed on the slide. It tapers at the ends to a sharp point. In length it varies from 4 to 20μ; in breadth it measures about 0.25μ. The spirals number from 6 to 26, averaging from 8 to 10. Not only are these spirals numerous but very narrow—not over about 1μ—regular and deep. Schaudinn, Hoffmann, Herxheimer, and others describe terminal flagella, but all observers are not agreed upon this point. When these are double they may really represent the commencement of a longitudinal fission. The spirochætæ may lie in clumps and occasionally form a felted mass. Löwenthal thinks that they are made up of several shorter (from 3 to 4μ) individuals, or possibly these may be degenerative forms. With Herxheimer, Mulzer, and others, he describes a granule or localised swelling between the middle and one terminal third, which may represent blepharoblast rather than nucleus. Some describe a terminal globular body, the real nature of which has not yet been ascertained. It may simply be a loop at the end of the spirochæta. Herxheimer has described other bodies in, on, or near the spirochæta. At first it was thought to possess a vibratile membrane attached throughout its whole length, but this idea is given up by most authors, with the exception of Dalous, the appearance being put down as artefact. The absence of such membrane along

[1] For a review of the literature on this subject, to January 1906, see author's papers in the *Lancet*, 1, 1906, pp. 663 and 746.

with the possession of flagella are held to be characters distinguishing it from other spirochætæ which commonly occur on ulcerated surfaces. Its motility is of three kinds: corkscrew, to and fro, and bending or lashing. In a film dried on the surface of glass the spirals may become stretched out owing to its becoming attached first at its extremities. By silver impregnation methods it has been demonstrated in sections of the liver in congenital syphilis and in the primary and secondary stages of the acquired disease. It lies in the capillaries of the papillæ of the corium, chiefly lying up against the endothelium or in the lumen. Thence it makes its way into the surrounding tissues and works up between the cells of the Malpighian layer to reach but not to penetrate the stratum corneum. With Giemsa's stain, it takes a rose-pink colour, or slightly lilac if the staining be too prolonged. It may also be demonstrated by "flagella" stains and impregnation methods, by gentian violet, carbol fuchsin, cresyl violet, Nile blue, etc. Schaudinn places spirochæta pallida with the protozoa rather than with spirillar bacteria, allying it to the trypanosome group.

The *spirochæta refringens* is generally acknowledged to be of saprophytic nature, and corresponds to the forms already described in normal smegma, in balano-posthitis, vulvo-vaginitis, the surface of ulcers about the genitals, or venereal warts, in the mouth secretions, on the teeth, tonsils, in ulcerated carcinomas, and probably also to the forms found in various forms of gangrene. It is thicker than spirochæta pallida, and distinctly refractile. It may have a marked corkscrew shape and motion when alive, but flattens out when dried in films, so as to have usually a wavy outline. The curves are flatter and longer than those of spirochæta pallida, measuring 1.5 to 2μ or more. It varies in length like the pallida, measuring most commonly from 8 to 10μ. It is usually described as having truncated ends, but often it has tapering ends or one end is sharp and the other blunt. It possesses a vibratile membrane but no flagella. It stains easily, quickly, and deeply by all methods, and with Giemsa's stain, which is the only differential one as yet known, it takes a purplish or violet colour. It occurs usually only on the surface of moist and ulcerated syphilitic lesions. Some describe it as invading the glands.

Demonstration of these parasites.—From the literature I have picked out the details as to 758 lesions examined by

different workers, and find that spirochæta pallida has been found most often in moist papules, mucous plaques, and condylomata genital and extra-genital—159 positive and 26 negative. Next come hard chancres, excised or scraped—150 positive and 55 negative; non-ulcerated papules—92 positive and 31 negative. Success has not been as frequent in films made from excised or punctured buboes—52 positive and 30 negative—and rarely has spirochæta pallida been found in roseolar blood —4 positive and 17 negative—or in the circulating blood—5 positive and 37 negative.

In lesions with moist or ulcerating surfaces the spirochæta pallida is commonly accompanied by the spirochæta refringens, and it is a very significant fact that most successes have been attained in such circumstances. From one's own experience the number of forms intermediate between the two, partaking to some extent now of the one, now of the other, is remarkable. It is sometimes extremely difficult, often impossible, to refer these forms to the one group or the other, and it is easy to imagine how readily one's percentage of successful demonstrations of the spirochæta pallida in such lesions might be raised by having regard solely to tinctorial or morphological characters. Frequently, in perfectly stained Giemsa preparations, I have seen forms, staining pink like spirochæta pallida, sometimes delicate but wavy or almost straight in outline like the spirochæta refringens, and, moreover, not in isolated examples, which might be explained as stretched spirochætæ pallidæ, but in very considerable numbers. Other forms stain deeply like spirochæta refringens, but they have a spiral outline like typical spirochætæ pallidæ. As a rule, these forms are thicker than pallida, and do not present so much difficulty as the other class of intermediate forms. Still, the fact remains that some authors figure such forms as spirochætæ pallidæ. As group characters one must take both tinctorial and morphological characters into consideration. The refinements of technique necessary to demonstrate flagella are too great to make their presence a point of great importance for the ordinary observer. When their presence is more widely confirmed, the methods of demonstration simplified, and staining results more constant, they might be of use in the diagnosis of the intermediate forms just referred to. It is regrettable that in condylomata and mucous plaques, the most infective lesions in the adult, these difficulties of diagnosis should be experienced.

Before making films from such lesions it is well thoroughly to cleanse the surface, as the better this is done the fewer saprophytic spirochætæ are met with. This refers also to hard sores, ulcerated papules, and rupioid or crusty syphilides. After cleansing the surface it should be scraped fairly deeply with a sharp spoon, the bleeding allowed in great measure to stop, and films (from six to eight) made from the material scraped away and from the bloody fluid which exudes. Some authors dilute these products, previous to making the films, with a drop or two of distilled water.

Hard chancres and *papules*, whether ulcerated or closed, may be excised, cut open from their under surface, and the expressed juice smeared over slides or treated by scraping, as in making films from moist or ulcerated lesions.

Blood. — Though several observers have found scanty spirochætæ in the blood during life, either by puncture of the skin or of the spleen, it is necessary in general to take a larger quantity of blood than is available by these means, and to treat it in the manner indicated first by Noeggerath and Staehelin. They adopted the method employed for the detection of trypanosomes in circulating blood. They take at least one cubic centimetre of blood from an arm vein, mix it with ten cubic centimetres of one-third per cent. acetic acid, centrifugalise and examine the deposit. One may remark that it is also possible, judging by analogy, from the fact that many trypanosomes, being of less specific gravity than the corpuscles, remain in the supernatant fluid, that similarly all the spirochætæ present may not be sent down by centrifugalising. Roseolar eruptions may be scraped or punctured, and the blood examined, or, following Levaditi and Petresco, small blisters may be raised over them by means of cantharides, and the fluid examined after centrifugalising. Several authors—*e.g.*, Bodin—report the failure of Levaditi's method in their hands. If, as some think, this lesion is caused by impaction of plugs of spirochætæ in the capillaries of the papillæ, they ought to be more easily demonstrable than they are at present.

Indurated buboes may be excised or punctured with a fairly stout hypodermic needle. After the usual antiseptic precautions, the gland is punctured, and the point of the needle kept near its surface, so as, if possible, to receive some lymph from the peripheral sinus. The gland meanwhile should be massaged

between the finger and thumb of the other hand. In view of the fact that some observers mix scrapings with a drop or two of distilled water to facilitate securing a thin film, it is probably not of importance to ensure the syringe being absolutely dry before using.

Congenital syphilis.—It is now a well-established fact that spirochæta pallida occurs very constantly in the blood and tissues of infants suffering from congenital syphilis. It has been found in the blood during life, and in the organs after death, especially in the liver, spleen, adrenals, kidneys, bone-marrow, lungs, and even once in the cerebro-spinal fluid. In the bullæ of pemphigus occurring in such infants, Levaditi, Salmon, Hoffmann, and others have found spirochætæ abundantly, often in clumps, apparently the result of agglutinative action. This is very important in consideration of the great infectivity and virulence of congenital syphilitic lesions, and is a fact greatly favouring an etiological relationship between spirochæta pallida and syphilis, as is also the fact that in tertiary syphilis, which is little or non-infective, most authors fail to find spirochæta pallida at all.

Fixation.—Most authors agree that the films, after drying in the air, should be fixed in absolute alcohol or alcohol-ether mixture for from ten minutes to twenty minutes, or in methyl alcohol for about five minutes. Schaudinn recommends that the films as soon as made should be exposed for two or three seconds to the vapour of osmic acid before drying and fixation in alcohol. For staining, Giemsa's medium is most used. It is related to Leishman's modified Romanowsky's stain, which gives as good results when one is expert in its use. Modifications of Pitfield's flagella stain give good results, but the spirochætæ appear thicker and coarser as well as darker than by other methods. This is still more evident in films and sections treated by silver impregnation methods. Schaudinn demonstrates the terminal flagella by Löffler's special method. Films are mounted in cedar oil or balsam.

The following points may be taken as axioms to be borne in mind :—(*a*) Saprophytic spirochætæ are always present on ulcerated lesions, on moist papules and mucous plaques. These are often difficult to distinguish from spirochæta pallida. (*b*) The more thorough the preliminary cleansing the fewer of these are seen. (*c*) Spirochæta pallida may occur somewhat deeply

in the lesions, and apparently in clumps. (*d*) On account of this, one must make six to eight films. (*e*) A little blood is usually an advantage in the film, as it assists in focussing, and, moreover, spirochætæ are often seen in apparent relationship to red blood corpuscles. (*f*) In the case of excised chancres and papules it is well to bear in mind that spirochæta pallida is not demonstrable after from six to ten hours, even though the tissue has been kept on ice. They soon degenerate.[1] (*g*) The films should be thin, and should be fixed as soon as possible. (*h*) When using Giemsa's method the nuclei of the leucocytes should be stained of a deep brownish purple, almost black, colour, otherwise the spirochætæ will not be seen. (*i*) Decolourisers such as alcohol or weak acetic acid should not be used, as they tend rapidly to discharge the stain from spirochæta pallida. This does not apply when the mordanting (flagella) methods are employed.

Cultivation.—Several attempts have been made to cultivate these spirochætæ, but so far without success. Judging, however, from the successful cultivation of trypanosomes, this should not be impossible. De Souza and Pereira used human blood with 5 per cent. each of citrate of sodium and chloride of sodium. Bertarelli and Volpino added gland juice to human blood with 50 per cent. of citrate of sodium, incubating at 27°, 30°, and 37° C.

Control observations.—A very large number of these have been made by numerous workers. Most agree that spirochæta pallida is not found in gonorrhœal pus and buboes, epididymitis, soft chancres and associated buboes, phagedænic ulcer, venereal warts, non-syphilitic ulcers of the glans, inflamed phimosis (except in one case in which a masked hard chancre was suspected), balanitis, and balanoposthitis; nor in smegma in man or woman, scabies, iodide rash, verruca juvenilis, scrofuloderma and lupus, phthisical sputum, furunculosis, acne, sycosis, dental suppuration, acute dermatitis, dermatitis herpetiformis, herpes zoster, eczema acute and chronic, eczema seborrhœicum, eczema papulo-crustosum, erythema exudativum multiforme, erythema nodosum, lichen ruber planus, psoriasis, pemphigus neonatorum, molluscum contagiosum, impetigo simplex and contagiosum, sarcoma and carcinoma of the penis and labia,

[1] The results of Levaditi's more recent researches show that this early degeneration does not always take place.—*Ann. de l'Inst. Past.*, i., 1906.

epithelioma cutis, carcinoma of the lip, the rectum, the neck, and the upper jaw, fibroma of the mamma, hypertrophy of the tonsils, Vincent's angina, throat mucus and mouth secretions, normal skin, organs of newly-born non-syphilitic children, and many other pathological and non-pathological tissues.

My own investigations embrace a comparatively small number of cases, the greater number of which have been obtained for me by Dr Alexander M. M'Intosh, clinical assistant in the lock wards of the Infirmary, who has also assisted me in the microscopical investigation of several of them. I am also indebted to him for furnishing me with histories of most of the cases.

CASE I.—A male, aged 18 years. He had had six weeks' illness. There were ulceration under the prepuce and gonorrhœa. On admission, in October 1905, there was a roseolar and papular rash over the whole of the body. There were œdema of the prepuce and a concealed hard chancre near the corona. The indurated groin glands were tender to the touch. Treatment consisted of mercurial inunctions; the prepuce was slit up, etc. The patient was readmitted at the end of November with gonorrhœal arthritis. Spirochæta pallida was found in the scrapings from the non-ulcerated papule on the thorax. The fluid from a groin gland gave a negative result. (Stain: Giemsa.)

CASE II.—A male, aged 25 years. He had had frequent connections, the last about three weeks before admisson. There were sores on the penis, and a rash appeared a few days later on the trunk and extremities (papular syphilide). There were mucous plaques over the pharynx. There were indurated buboes. On examination, fluid from the glands gave a negative result. In scrapings from the hard chancre, spirochæta pallida was present along with spirochæta refringens. Scraping from the dry papulo-squamous syphilide proved negative. (Stain: Giemsa.)

CASE III.—A male, aged 22 years. Infection occurred about twelve months before admission. There were typical peri-anal condylomata. Scrapings from these showed numerous spirochætæ refringentes and many intermediate forms, many delicate, staining pink with Giemsa, but straight or wavy in contour. Some forms were thin, with narrow spirals, but staining of a bluish or purplish colour.

CASE IV.—A male, aged 19 years. There was a two months' history. There was a diffuse macular and papular rash over the face and the body. The prepuce was œdematous and there was a concealed hard chancre. The glands were not typical. Treatment consisted in circumcision and mercurial inunctions. Films made from the excised chancre contained a few spirochætæ pallidæ and a peculiar flagellated body. The films were examined by both Dr M'Intosh and myself. The body stains a brownish colour with Giemsa, and in it a clear spot resembling a vacuole was readily distinguished. It had at least six angles, and to these whip-like appendages resembling spirochæta pallida in tinctorial and morphological characters were attached. In the case of two of these the thickness was greatest near the body and gradually lessened as one passed out. The appearance was evidently not an artefact, and the staining of the central mass was not like that of red blood corpuscles in the film. As to its nature one can make no suggestion, and I have not seen another body like it. It may possibly be a red blood corpuscle to which spirochætæ are attached, and the hæmoglobin of which is diffusing along their proximal portions. If it is of importance in connection with spirochæta pallida it is curious that no other observer has seen an exactly similar object. This very fact tends to make one very sceptical as to its importance. Still, it is necessary to put the fact on record and microphotographs of it are reproduced (Figs. 5 and 7). In the same cases numerous spirochætæ pallidæ were found in the scrapings from a closed papular syphilide. (Stains: Giemsa and modified Pitfield's flagella.)

CASE V.—A male, aged 22 years. Infection occurred seven weeks before admission. There were a concealed hard chancre and a diffuse and roseolar rash. There were typical indurated groin glands. Treatment consisted in circumcision and mercurial injections. On examination scrapings from the macular eruption proved negative. Films from the excised hard chancre contained numerous spirochætæ pallidæ. (Stains: Giemsa, modified Pitfield's flagella, and carbolic gentian violet.)

CASE VI.—A male, aged 19 years. Infection occurred from five to eight weeks before admission. There was a hard chancre on the prepuce and a macular eruption; the indurated groin glands were typical. Circumcision was performed and

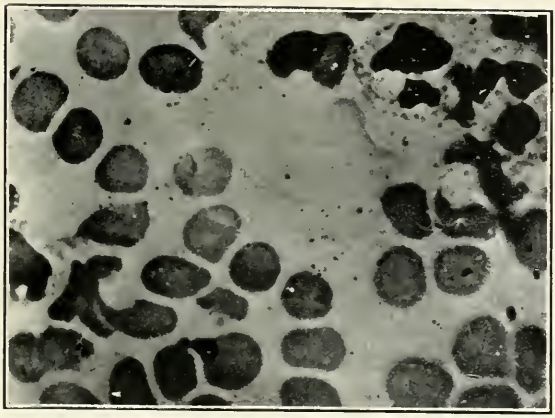

FIG. 1.—Spirochæta pallida in apparent relation to a red blood corpuscle. Stained with Giemsa's solution. The organism appears in the centre of the field, sloping downwards from left to right.

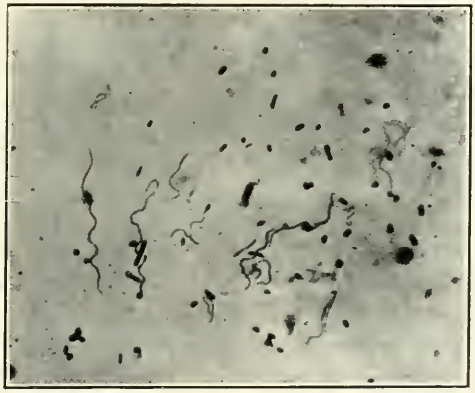

FIG. 3.—Film from condyloma latum showing numerous forms, mostly intermediate between spirochæta pallida and spirochæta refringens. Numerous bacteria. Stained with Giemsa's solution.

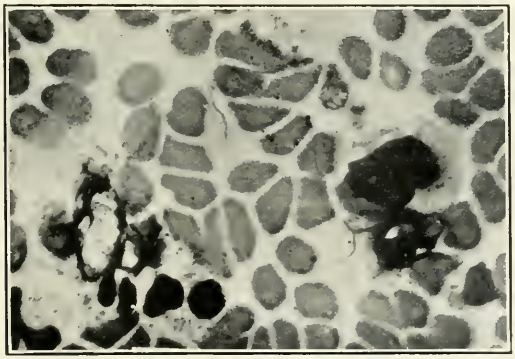

FIG. 4.—Spirochæta refringens. Two examples scraped from a syphilitic hard sore. Stained with Giemsa's solution.

PLATE IV.—TO ILLUSTRATE DR SHENNAN'S ARTICLE.

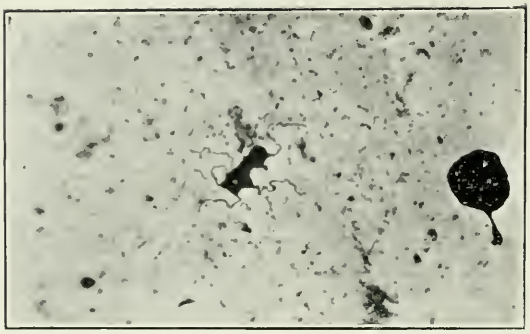

FIG. 5.—Peculiar flagellated body found in film made from fluid expressed from the cut surface of a hard chancre. Stained with Giemsa's solution. × circ. 1200.

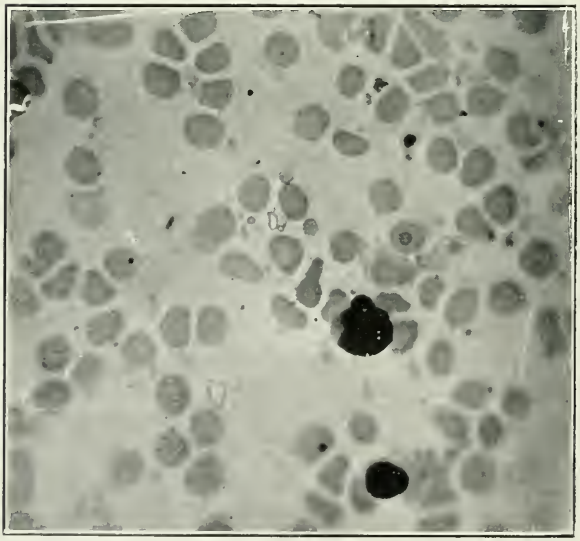

FIG. 6.—Peculiar loops and roundish bodies found not uncommonly in smears from lymph glands. Loops staining with Giemsa's solution red. Roundish bodies staining with Giemsa's solution blue.

PLATE V.—TO ILLUSTRATE DR SHENNAN'S ARTICLE

FIG. 7.—*a-i* × about 1000, *k* × about 1200. *a*, Spirochæta pallida—a_1, in relation to red blood corpuscles; a_2, crossing an oblong body (particle of chromatin?) and apparently attached to one of the angles; a_3, crossing red blood corpuscles showing apparent thickening. *b*, Spirochæta refringens. *c*, Fusiform body like baccilus fusiformis, frequently seen in the ulcerated lesions. *d*, Intermediate forms from condyloma latum. *e*, Artefacts, drawn-out nuclei, etc. *f*, A thin filament of fibrin (?) simulating spirochæta pallida. *g*, Wrinkled margin of deformed red blood corpuscle which might be mistaken for spirochæta pallida encircling it. *h*, Peculiar loop-shaped bodies staining bright pink with Giemsa's solution found in several of the films from the glands. Their relationship to the spirochætæ though possible is not clear. *i*, Rounded bodies (Winckler's?) in gland fluid. They stain a pale blue with Giemsa's stain. *k*, Peculiar body found once in hard chancre. Note the fusiform thickening seen in the course of the flagella-like extensions.

PLATE VI.—TO ILLUSTRATE DR SHENNAN'S ARTICLE.

mercurial injections were given. Scrapings from the macular eruption proved negative. Films from the excised hard chancre contained numerous spirochætæ pallidæ. (Stain : Giemsa.)

CASE VII.—A male, aged 38 years. A hard chancre appeared five weeks before admission. There was a typical hard chancre on the dorsum of the penis. There were typical groin glands and papular syphilide. This was one of the first cases I examined. I made only three films from the chancre and could not find spirochætæ. (Stain : Geimsa.)

CASE VIII.—A male, aged 23 years. Thirteen days after the last connection (six and three weeks before admission) an ulcer appeared on the right side of the glans and rapidly enlarged, eating into the glans deeply. There was no glandular enlargement or induration. There was no rash. The ulcer healed rapidly under treatment with lotio nigra. Films from the ulcer contained numerous spirochætæ refringentes. There were no spirochætæ corresponding to spirochæta pallida. (Stain : Giemsa.)

CASE IX.—A female, aged 32 years. There was a doubtful ulcer at the fourchette. Films made from this contained numerous bacteria and spiral forms, most corresponding morphologically to spirochæta refringens, but a few delicate with narrow spirals like spirochæta pallida. On account of this result I asked Dr M'Intosh to watch this patient carefully. About a week later a typical macular rash appeared on the thighs and thorax, the primary sore becoming quite typical. (Stain : Giemsa.)

CASE X.—A male, aged about 24 years. He had had syphilis seven or eight years ago. Films made from scrapings of the sores contained numerous intermediate forms, some of which were apparently spirochætæ pallidæ and numerous forms corresponding to spirochæta refringens.

CASE XI.—A female, aged 39 years. There were condyloma, papular rash, and indolent buboes in the groin and behind the sterno-mastoid. Six films were brought to me which had been made from the condyloma. No spirochætæ pallidæ were found in the six films.

CASE XII.—A male, aged 22 years. The last connection was eleven days before admission. He was discharged on the ninth day. He had œdema of the prepuce, phimosis, and concealed soft chancre. There was no enlargement of the

glands and no rash. Circumcision was performed, removing the sore. Films showed no spirochætæ like pallida.

CASE XIII.—A male, aged 24 years. The patient was infected with gonorrhœa and sores about eight months before admission. Now he had induration at the preputial orifice. There was a diffuse macular rash on the body with a few papules on the penis and pubes. Typical indolent buboes were present in the groin and behind the sterno-mastoid. The hard chancre was excised. Films were made from scrapings from the surface and fluid from the cut surface. In eight films no spirochætæ pallidæ were discovered.

CASE XIV.—A male, aged 35 years. There was a three and a half months' history. There was a papulo-squamous syphilide on the legs and trunk, which was drying up under treatment. He was twice examined with a negative result.

CASE XV.—A male, aged 20 years. There was a three months' history. He had perianal condylomata, which were now drying up under local and general treatment. A few spirochætæ refringentes were found but no spirochætæ pallidæ.

CASE XVI.—A male, aged 24 years. Infection occurred ten months before admission. A sore formed which never completely healed. Connection meanwhile was almost fortnightly. Three weeks before admission the sore broke down extensively and became indurated and indolent. The glands were enlarged and hard. There was a papular rash. From fluid from the glands films were made. No spirochætæ pallidæ were found, but a few peculiar tailed bodies like spermatozoa. These were possibly drawn-out nuclei of leucocytes.

CASE XVII.—A male, aged 19 years. Infection occurred four weeks prior to admission. On admission the patient had a concealed hard chancre. Circumcision was performed. Four weeks later (December 1905) there were a dry parchment sore on the glans which was healing, and indurated groin glands. Scrapings from the sore on the glans showed presence of spirochæta refringens and some intermediate forms, some of which approximated in form but not in colour to spirochæta pallida. Some of them had a small dark body attached at one end. Fluid from the groin glands contained no demonstrable spirochætæ but numerous flattened or oval loop-like structures, staining bright pink with Giemsa's solution, were seen (Figs.

6 and 7). Some of these were shaped like the "eye" of the "hook and eye" of the dressmaker. In the same case there were rounded bodies varying in size from 1μ to 4μ, staining a delicate blue with Giemsa, sometimes single, sometimes in clusters, as if they had been contained within a phagocytic cell which had broken up in the process of making the film. These were probably products of the degeneration of cells. They are possibly analogous to the bodies described by Winckler.

CASE XVIII.—A male, aged 20 years. There was a two months' history. He had a general rupial eruption over the whole of the body resembling impetigo at parts. Scrapings from the bases of these lesions and fluid expressed from them failed to show presence of spirochæta pallida.

CASE XIX.—A male, aged 22 years. There was a thirteen months' history. Treatment had been irregular. There were perianal condylomata. Films were made from the condylomata, but no spirochætæ pallidæ could be detected.

CASE XX.—The hard chancre of another male was examined and numerous spirochætæ pallidæ were discovered. No history of the case is now obtainable.

CASE XXI.—A male, aged 28 years, suffering from tabes. Symptoms began three months after infection with syphilis three years ago. Spinal fluid was removed by lumbar puncture. No spirochætæ were found. On cultivation a pure culture of streptococci developed in one tube.

Of these twenty-one cases, two (VIII. and XII.) were not syphilitic; another (XXI.) was a case of tabes dorsalis three years after infection. Excluding these there are left eighteen cases showing twenty-five lesions examined. Out of the eighteen cases six showed the presence of undoubted spirochæta pallida. In three others spirochætæ were found which could not with certainty be placed either with spirochæta pallida or spirochæta refringens. One case (VII.) was examined when I had little experience in looking for spirochæta pallida. In one case (IX.) the possibilities are in favour of the bodies seen being spirochætæ pallidæ. In two cases (XIV. and XV.) the lesions were drying up under treatment. The results were quite negative in the remaining five cases.

As regards the lesions five hard chancres showed positive results; in two they were doubtful and in three they were

negative. Two non-ulcerated papules gave positive results; two dry papulo-squamous syphilides gave negative results; and two roseolar rashes also were negative. Four condylomata were examined; three were negative and one was doubtful. Four glands were examined and in none could undoubted spirochætæ pallidæ be found. One rupioid syphilide was examined with a negative result. Typical spirochætæ pallidæ were found only in hard chancres and in closed papules, *i.e.* in typical syphilitic lesions.

Conclusions.

It is astonishing with what unanimity this discovery has been accepted on the Continent, an occurrence quite unprecedented in discoveries of similar importance, but there is much to support the relationship of spirochæta pallida to syphilis. The most important facts in favour are its occurrence in non-ulcerated lesions, such as papules and buboes far away from the primary sore; its occurrence in the circulating blood and splenic blood in acquired syphilis, its occurrence specially in virulent infective lesions, and its absence from non-infective tertiary lesions; its presence in the depths of superficial lesions where it is stated to be unaccompanied by other organisms, and its definite localisation in the corium—if absolutely confirmed; its presence in congenitally syphilitic infants in great numbers, and its occurrence—according to most observers—only in primary and secondary syphilitic lesions and in congenital syphilis—never in non-syphilitic diseases. Against it is its frequent scanty numbers, films in many of the reported successful cases revealing the presence of only one or two spirochætæ, after repeated and prolonged search, the numbers thus frequently bearing no relationship to the severity of the tissue reactive changes, which is, as Siebert points out, without analogy in the domain of pathology; the possibility, which is by no means disproved, that various organisms may invade glands, penetrating from a superficial ulcer; the difficulty of diagnosis in the case of the numerous intermediate forms which are found so commonly in such highly infective lesions as the mucous plaque, etc.; the claim, still strongly upheld by some, that forms indistinguishable from spirochæta pallida are found in non-specific lesions. The last point, of course, even though

it were granted, does not necessarily invalidate a causative relationship to syphilis.

In conclusion, one may say that while there is a very considerable probability that spirochæta pallida bears an etiological relationship to syphilis we require to know more about spirochætæ in general, and to have better methods of distinguishing the many forms intermediate between the typical pallida and the typical refringens, before we can unreservedly accept this as a completely proved fact in pathology. One can say very little as yet with regard to the peculiar loop-like and rounded bodies found in syphilitic glands, and to such bodies as the flagellated structure I have described in a film from a hard chancre.

Meeting V.—February 7th, 1906

Dr J. O. Affleck, *President, in the Chair*

I. Election of Members

John A. Gunn, M.B., B.Sc., Materia Medica Department, University of Edinburgh, and Fred. Dihtmar, M.D., D.P.H., 26 Heriot Row, were elected Ordinary Members of the Society.

II. Exhibition of Patients

1. In the absence of *Dr W. Allan Jamieson*, *Dr Norman Walker* exhibited a case of EXTRA-GENITAL CHANCRE.

A young woman, who had presented herself at the Royal Infirmary a fortnight previously with a button-like growth on the right side of the chin. This was quite round, three-quarters of an inch in diameter, and its convex surface rising to half an inch above the skin at its highest part. It was a dull pinkish red, bore some crusts, and had a hard stony feel. The submaxillary and cervical glands on the right side were enlarged. There was a small reddened area on the right side of the arch of the fauces. On the back of the neck, bosom, and front of the thighs was a faint roseolar or macular rash. This, she naïvely observed, was distinctly brighter when the parts were exposed to cold, thus unconsciously aiding the diagnosis. The commencement dated back two months, and the source of infection was obscure, though she admitted having been kissed at games. When shown, the roseolar rash had faded, as a result of mercurial treatment.

2. *Dr Norman Walker* exhibited a boy, aged 5. Six months previously he was brought to the Royal Infirmary on account of a number of black spots on his cheek. These have increased in size and number, and some on the ear have taken on a warty character. Dr Walker had never seen a similar case, and was doubtful whether it was a unilateral xeroderma pigmentosum or an unusual example of the malignant melanoma.

III. Exhibition of Specimens

Mr J. M. Cotterill exhibited—

(*a*) Fibrosarcoma of eighth cranial nerve, removed from the cerebello-pontine angle. The tumour was reached by resecting a considerable portion of the left lobe of the cerebellum.

(*b*) and (*c*) Senile hypertrophy of the prostate, from men aged 76 and 80. The supra-pubic route was used, and the wounds were healed in three weeks.

(*d*) Scirrhus of the prostate, from a patient aged 71. Symptoms for 2½ years. Gland very adherent to right vesicula seminalis and ejaculatory ducts, which were in part removed. Water passed per urethram on the thirteenth day after operation. The supra-pubic method was used.

IV. Original Communications

1. RECTAL ALIMENTATION

By Francis D. Boyd, M.D., C.M.G., F.R.C.P. Ed., Assistant-Physician, Royal Infirmary; Physician, Deaconess Hospital, Edinburgh

(From the Research Laboratory of the Royal College of Physicians, Edinburgh)

Rectal alimentation is so universally in use in clinical medicine that it is surprising that at the present day we have not satisfactory and accurate data on which to gauge its value; for while a good deal of work has been done on the subject, much of it is unsatisfactory in the light of modern methods. Though rectal feeding dates back to the days of Galen, Voit and Bauer seem to have been the first to carry out exact observations. It is, however, the work of Leube and of Ewald which has been accepted as the basis on which most statements have been made as to its value. As the results obtained by these observers seemed to us unconvincing in certain re-

spects, the following observations were undertaken with a view to gauging, if possible, the influence which this mode of alimentation has upon the metabolic processes of the body.

Methods.—The observations were carried out on patients suffering from gastric ulcer, when a period of complete gastric rest seemed necessary as a therapeutic measure. The patient was prepared for treatment by the administration of a brisk purge along with a dose of charcoal. When the charcoal appeared in the stool, it was considered that the alimentary canal had been emptied. Between the giving of the aperient and the commencement of rectal feeding, no food was allowed by the mouth; the patient was kept absolutely at rest in bed. The rectal nourishment selected was given in the form of an enema every six hours, four being thus given in the twenty-four hours. At the end of every period of twenty-four hours the bladder was emptied, and the rectum washed out with a simple water enema. The nutrient enemata were made up in bulk by a firm of wholesale chemists, divided by weight, and cinnamon water or chloroform water used as a preservative. When chloroform was used, it was dissipated before administration by placing the uncorked bottle containing the injection in warm water for an hour. Two sample bottles of each of the different series were taken for estimation of the constituents (nitrogen, fat, and sugar) of the enema. Before administration, in the cases in which eggs were used, the enemata were pancreatised. When milk was used it was pancreatised and sterilised, aud a sample taken for estimation of the constituents. Milk sugar was inverted before estimation by Fehling's method. The urine and bowel contents were carefully collected. The nitrogen estimations were carried out by Kjeldahl's method. The nitrogen of the bowel contents and of the enemata, except in the first observation, was estimated on the dried material, two estimations of each being made and an average obtained. If there was any distinct discrepancy in the two results, the estimation was repeated.

The fats were estimated by weight after extraction with ether in Soxhlet's apparatus.

To determine the sugar, a sample of the bowel contents was taken, animal charcoal added, and the whole heated to precipitate albumen. Filtration gave a colourless fluid, in which the sugar could be estimated by Fehling's method.

The patient's weight was determined every third day.

Much difficulty was caused with female patients by the incident of menstruation. Rectal feeding seems to have a very marked influence in inducing menstruation even when the period is not due. A large number of our observations broke down owing to this cause, and are not used as data in this paper.

Observation I.—A fairly well nourished young woman. Symptoms of gastric ulcer lasting over several years. Put on rectal feeding after brisk purgation and twenty-four hours' starvation.

Rectal feeding for six days.

Enema: Two eggs.
Dextrose.
10 c.c. Cod liver oil.
Normal saline solution.

Enemata well retained.

After six days, all pain having disappeared, milk was given by the mouth. Patient appeared well and comfortable during period of rectal feeding. Recovery was uninterrupted.

OBSERVATION I.

Day.	Enemata.			Excretion.				Weight of Patient in Kilogrammes.
	Nitrogen.	Fat.	Sugar.	N. of Fæces.	N. of Urine.	Fat.	Sugar.	
I.	11.56	104.37	47.75	10.73	4.66	67.06		51.529
II.	11.56	104.37	47.75	12.39	3.38	78.00	3.22	
III.	11.37	102.88	47.75	8.97	3.64	51.03	2.68	
IV.	11.37	102.88	47.75	8.04	2.63	38.87	4.00	
V.	11.37	102.88	47.75	8.41	2.66	46.28	8.08	
VI.	11.37	102.88	47.75	10.80	2.69	63.90	5.71	49.259
Totals	68.60	620.26	286.50	59.34	19.66	345.14	23.69	Loss in weight 2.27 kilos.

Day.	Nitrogen absorbed.	Nitrogen of Urine.	Nitrogen from Tissues.	N. Value as Proteid of Tissues.
I.	0.83	4.66	3.83	23.93
II.	−0.83	3.38	4.21	26.31
III.	2.40	3.64	1.24	7.75
IV.	3.33	2.63	−0.70	−4.37
V.	2.96	2.66	−0.30	−1.87
VI.	0.57	2.69	2.12	13.25
Totals	9.26	19.66	10.40	65.00

Caloric value of material metabolised per diem as average over period :—

Proteid.	Calories.	Fat.	Calories.	Sugar.	Calories.	Total Calories.
9.62	39.46	45.85	426.40	43.80	179.58	645

Observation II.—Well nourished young woman, aged 20. Chronic gastric ulcer with slight gastric dilatation.

Enema : 2 eggs.
20 grm. dextrose.
NaCl.
Chloroform water to 300 c.c.

Enemata fairly well retained. On third day of rectal feeding some headache and sickness but no vomiting. After period of gastric rest, convalescence uninterrupted.

Observation II.

Day.	Enemata.			Excretion.				Weight of Patient in Kilogrammes.
	Nitrogen.	Fat.	Sugar.	N. of Fæces.	N. of Urine.	Fat.	Sugar.	
I.	7.32	53.07	38	4.87	6.05	21.18	0	46.535
II.	7.32	53.07	38	3.58	4.48	15.38	0	
III.	7.32	53.07	38	5.93	6.55	24.83	0	46.215
IV.	7.32	53.07	38	6.21	6.30	27.85	0	
V.	5.36	36.20	38	6.21	6.30	27.85	0	
VI.	5.36	36.20	38	6.61	3.36	24.33	0	45.0595
Totals	40.00	284.68	228	33.41	33.04	141.42	0	Loss in weight 1.4755 kilos.

Day.	Nitrogen absorbed.	Nitrogen of Urine.	Nitrogen from Tissues.	N. Value as Proteid of Tissues.
I.	2.45	6.05	3.60	22.50
II.	3.74	4.48	0.74	4.62
III.	1.39	6.55	5.16	32.25
IV.	1.11	6.30	5.19	32.43
V.	−0.85	6.30	7.15	44.68
VI.	−1.25	3.36	4.61	28.81
Totals	6.59	33.04	26.45	165.29

Caloric value of material metabolised per diem as average over period :—

Proteid.	Calories.	Fat.	Calories.	Sugar.	Calories.	Total Calories.
6.81	27.93	23.87	221.99	38	155.8	405

Observation III.—Fairly well nourished young woman, aged 30. Relapsed gastric ulcer.

Enema : Eggs.
Dextrose.
Normal saline solution.

Enemata fairly well retained. On two occasions urine and bowel contents were mixed. The urinary nitrogen on these days was got approximately, by averaging the nitrogen of the fæces on the three previous days and deducting it from the total nitrogen of the day in question.

The patient passed through a satisfactory convalescence.

OBSERVATION III.

Day.	Enemata.			Excretion.				Weight of Patient in Kilogrammes.
	Nitrogen.	Fat.	Sugar.	N. of Fæces.	N. of Urine.	Fat.	Sugar.	
I.	8.36	41.09	68.16	6.57	2.11	20.78	0	48.9185
II.	8.36	41.09	68.16	4.86	2.29	21.55	0	
III.	8.36	41.09	68.16	6.64	3.75	26.42	0	
IV.	7.48	39.61	57.12	6.02	2.22	32.05	0	546.6485
V.	7.48	39.61	57.12	6.31	3.11	24.51	0	
VI.	7.48	39.61	57.12	6.34	3.18	19.60	0	
VII.	7.48	39.61	57.12	6.33	2.15	35.55	0	46.081
Totals	55.00	281.71	432.96	43.07	18.81	180.46	0	Loss in weight 2.8375 kilos.

Day.	Nitrogen absorbed.	Nitrogen of Urine.	Nitrogen from Tissues.	N. Value as Proteid of Tissues.
I.	1.79	2.11	0.32	2.00
II.	3.50	2.29	−1.21	−7.56
III.	1.72	3.75	2.03	12.68
IV.	1.46	2.22	0.76	4.75
V.	1.17	3.11	1.94	12.12
VI.	1.14	3.18	2.04	12.75
VII.	1.15	2.15	1.00	6.25
Totals.	11.93	18.81	6.88	42.99

Caloric value of material metabolised per diem as average over period :—

Proteid.	Calories.	Fat.	Calories.	Sugar.	Calories.	Total Calories.
10.62	43.56	14.46	134.47	61.85	253.58	431

Observation IV.—Young woman, aged 25. Poorly nourished. Somewhat nervous and emotional. Signs and symptoms of chronic gastric ulcer.

Enema : 2 eggs.
Dextrose.
NaCl.

Enemata well retained, but complaint of considerable gastric pain after injection.

After rectal feeding, progress was slow, gastric pain being set up whenever food was given. After a prolonged restricted diet, digestive symptoms were markedly ameliorated.

Observation IV.

Day.	Enemata.			Excretion.				Weight of Patient in Kilogrammes.
	Nitrogen.	Fat.	Sugar.	N. of Fæces.	N. of Urine.	Fat.	Sugar.	
I.	7.48	39.61	57.12	7.46	6.72	35.72	10.10	50.394
II.	7.48	39.61	57.12	6.04	5.35	37.31	0	
III.	7.48	39.61	57.12	6.26	6.18	31.33	13.6	
IV.	7.48	39.61	57.12	8.10	6.22	35.70	5.08	
V.	7.48	39.61	57.12	7.10	6.16	41.15	16.76	
VI.	7.48	39.61	57.12	5.36	6.61	29.16	0	
VII.	7.48	39.61	57.12	7.71	6.97	28.73	0	46.762
Totals	52.36	277.27	399.84	48.03	44.21	239.10	45.54	Loss in weight 3.632 kilos.

Day.	Nitrogen absorbed.	Nitrogen of Urine.	Nitrogen from Tissues.	N. Value as Proteid of Tissues.
I.	0.02	6.72	6.70	41.87
II.	1.44	5.35	3.91	24.43
III.	1.22	6.18	4.96	31.00
IV.	−0.62	6.22	6.84	42.75
V.	0.38	6.16	5.78	36.12
VI.	2.12	6.61	4.49	28.06
VII.	−0.23	6.97	7.20	45.00
Totals	4.33	44.21	39.88	249.23

Caloric value of material metabolised per diem as average over period :—

Proteid.	Calories.	Fat.	Calories.	Sugar.	Calories.	Total Calories.
3.86	15.63	5.45	50.68	50.61	207.50	273

Observation V.—Very well nourished girl, 17 years of age. Somewhat chlorotic. Gastric ulcer.

Enema: 200 c.c. milk.
White of one egg.
Dextrose.
1 grm. NaCl.
Water to 300 c.c.
Peptonised, sterilised.

Enemata well retained. Recovery uninterrupted.

Observation V.

Day.	Enemata.			Excretion.				Weight of Patient in Kilogrammes.
	Nitrogen.	Fat.	Sugar.	N. of Fæces.	N. of Urine.	Fat.	Sugar.	
I.	4.87	9.53	91.41	2.42	4.23	8.30	16.66	44.946
II.	4.87	9.53	91.41	3.46	6.41	10.86	5.41	
III.	4.87	9.53	91.41	3.09	6.92	11.60	12.14	
IV.	4.87	9.53	91.41	3.77	8.20	9.44	6.54	
V.	4.87	9.53	91.41	4.61	8.43	18.74	3.33	
VI.	4.87	9.53	91.41	3.38	7.84	13.14	2.90	
VII.	3.65	7.14	68.56	2.44	6.10	10.09	2.31	40.860
Totals	32.87	64.32	617.02	23.17	48.13	82.17	49.29	Loss in weight 40.086 kilos.

Day.	Nitrogen absorbed.	Nitrogen of Urine.	Nitrogen from Tissues.	N. Value as Proteid of Tissues.
I.	2.45	4.23	1.78	11.12
II.	1.41	6.41	5.00	31.25
III.	1.78	6.92	5.14	32.12
IV.	1.10	8.20	7.10	44.37
V.	0.26	8.43	8.17	51.06
VI.	1.49	7.84	6.35	39.68
VII.	1.21	6.10	4.89	30.56
Totals	9.70	48.13	38.43	240.16

Caloric value of material metabolised per diem as average over period :—

Proteid.	Calories.	Fat.	Calories.	Sugar.	Calories.	Total Calories.
8.65	35.36	(-2.55	-23.71)	81.10	332.51	344

Observation VI.—Fairly well nourished young woman. Somewhat anæmic. Symptoms of gastric ulcer for over a year.

Enema : 200 c.c. milk.
Dextrose.
White of one egg.
1 grm. NaCl.
Water to 300 c.c.
Peptonised and sterilised.

Enemata very well retained. Felt very well during rectal feeding. Recovery uninterrupted.

Observation VI.

Day.	Enemata.			Excretion.				Weight of Patient in Kilogrammes.
	Nitrogen.	Fat.	Sugar.	N. of Fæces.	N. of Urine.	Fat.	Sugar.	
I.	4.90	14.35	39.08	1.71	8.29	5.83	1.00	45.4
II.	4.90	14.35	39.08	1.14	9.83	5.23	0	
III.	4.90	14.35	39.08	2.84	9.69	10.31	1.97	
IV.	4.90	14.35	39.08	2.90	9.32	11.22	2.50	42.222
V.	4.90	14.35	39.08	3.40	8.01	12.82	3.63	
VI.	4.90	14.35	39.08	3.81	7.11	16.31	2.91	
VII.	4.90	14.35	39.08	2.90	4.62	14.36	2.79	39.952
Totals	34.30	100.45	273.56	18.70	56.87	76.14	14.80	Loss in weight 5.448 kilos.

Day.	Nitrogen absorbed.	Nitrogen of Urine.	Nitrogen from Tissues.	N. Value as Proteid of Tissues.
I.	3.19	8.29	5.10	31.87
II.	3.76	9.83	6.07	37.93
III.	2.06	9.69	7.63	47.68
IV.	2.00	9.32	7.32	45.75
V.	1.50	8.01	6.51	40.68
VI.	1.09	7.11	6.02	37.62
VII.	2.00	4.62	2.62	16.37
Totals	15.60	56.87	41.27	257.90

Caloric value of material metabolised per diem as average over period :—

Proteid.	Calories.	Fat.	Calories.	Sugar.	Calories.	Total Calories.
13.87	56.88	3.47	32.27	36.96	151.53	240

The Absorption and Metabolism of Albumin.

Voit and Bauer seem to have been the first to carry out observations on the nitrogenous metabolism of rectal feeding ; working with dogs they found that the absorption of albumin was greatly aided by the addition of salt.

Eichorst repeated these experiments and obtained similar results.

Leube in 1872 published a paper on meat and pancreas enemata, by which he claimed to get an absorption equivalent to 500 calories per diem.

Ewald in 1887 contributed a paper on egg and peptone enemata which has been accepted as the basis on which most modern statements are made as to the value of rectal feeding. Exception may be taken to Ewald's methods, for phenolpthalin was used as an indicator in the nitrogenous estimations, and it is now universally admitted that phenolpthalin is useless as an indicator when dealing with ammonia. The results in this paper are expressed in the form of a graphic chart which, though at the first glance appearing very convincing, is in reality exceedingly complicated add difficult to follow. When the data are carefully analysed and the figures written down, they appear insufficient to form a metabolic profit and loss account, and the reader is unable from the data given to submit the author's claims to criticism. Judging by the results of other observers, the absorption of proteid in the cases quoted by Ewald, if it is to be accepted, was quite exceptional.

Armin Huber, working at the nutritive value of egg injections, gives observations on three patients. The patients were kept on a fixed diet throughout the observation. For three or four day periods, enemata of emulsified egg or egg and salt were given in addition to the diet. The periods during which the enemata were given alternated with periods of food by the mouth alone. The results given by Huber are very variable —so variable that one is driven to conclude that there must be considerable experimental error in many of the figures. In Observation A, Period V., with enema of egg and salt, there is a nitrogen balance of + 3.68 ; while in Period VII., with the same injections, there is a balance of + 23.05. In Observation C, a man aged 51, one finds even greater apparent discrepancies. In Period III., when *no* enemata were given, there is a positive

balance of 3.83. In Period IV., when eggs and salt were given per enema, a positive balance of only 2.38, while the patient is still losing weight. In Period V., with no enema, a negative balance of 5.92. In Period VI., a positive balance of 7.74, while the weight of the patient remained stationary. Such results are eminently unsatisfactory.

Ehrstrom obtained relatively good results with the use of milk, grape sugar and proton—a soda-casein preparation. His patient absorbed daily 3.92 gr. of nitrogen, and was not thereby kept in nitrogenous balance ; but that is not surprising, as the total nourishment given was only equal to 700 calories.

Bial claims to have got favourable results with the use of Witte's peptone ; he made the observations on himself. There was first a period of starvation, followed by a one-day period during which three enemata of 250 c.c. 10 per cent. solution of peptone were taken ; to one of these alcohol was added. Bial claims to have got an absorption of 50.5 per cent. of peptone, and 66 per cent. in the case when alcohol was added. Eichorst's observation on a dog can scarcely be considered reliable, as the dog was suffering from glycosuria and albuminuria.

TABLE I.

Table to show proteid and nitrogen of food and proteid and nitrogen absorbed, with caloric value of absorption as average per diem :—

	Enema.		Absorption.[1]		
Observation.	Proteid.	Nitrogen.	Proteid.	Nitrogen.	Caloric Value.
I.	71.43	11.43	9.52	1.54	39
II.	41.62	6.66	6.87	1.098	27
III.	49.06	7.85	10.52	1.7	43
IV.	46.75	7.48	3.86	0.618	15
V.	29.71	4.69	8.62	1.38	35
VI.	30.6	4.9	13.87	2.22	56

Plantenga found very small absorption of albumin even when salt solution was added, and Markwald comes to the

[1] Caspari, in a recent physiological study on Vegetarianism (*Pflügers Archiv*, Bd. cix., 1905, S. 533), calculates that the amount of nitrogen contained in the digestive secretions equals 0.38 grammes per diem. If this observation be correct, and if it applies to cases under rectal alimentation, it would increase the nitrogen absorption by that figure. The tables have not been calculated on this basis, as we have no sufficient evidence of the digestive secretions under rectal alimentation.

same conclusion. Plantenga, in a second communication, investigated the absorption of somatose from the lower bowel of the dog. He found in his observations an absorption averaging from 10.8 to 24.1 per cent.; a very poor result.

Edsall and Miller studied two patients very fully. They worked with milk and eggs, and found that the nitrogen absorptions daily equalled, in the first case, 3.041 grm. nitrogen, or 19 grm. proteid, while in the second it equalled 3.809 grm. nitrogen, or 23.816 grm. proteid. There seems thus a consensus of opinion that albumin is but poorly absorbed even when predigested and when salt is added. This conclusion is the more remarkable when we consider how largely the white of egg is used in rectal feeding.

Our observations are given in Table I.

In Observations I. to IV. inclusive, considerable amounts of proteid were used (two eggs in each injection). In observations V. and VI. the white of one egg was used along with milk. In all the observations the amount of proteid absorbed is remarkably small. In Observation IV., a poorly nourished, somewhat nervous subject, the amount absorbed is particularly small. In Observations V. and VI., where a smaller amount of proteid was given in the injection, the absorption was just as good as when a larger amount was given. In fact, in Observation VI., the absorption of proteid was better than in any of the preceding cases, and this notwithstanding the fact that milk was used in the enema, and milk is generally considered, except by Aldor, to be a food-stuff very poorly assimilated by the bowel in rectal feeding.

TABLE I.A.

To show absorption of nitrogen per kilo. of body weight:—

Observation.	N. absorbed.	Body Weight.	N. absorbed per Kilo.
I.	1.54 grm.	51.5	0.029 grm.
II.	1.09 „	46.5	0.0234 „
III.	1.7 „	48.9	0.035 „
IV.	0.61 „	50	0.012 „
V.	1.38 „	45	0.03 „
VI.	2.22 „	45	0.049 „
Average	1.42 „		0.0297 „

When the absorption of nitrogen is considered as per kilo. of body weight per diem, it is seen to be very small indeed. It is remarkable, when one looks into the literature of the subject, on what a small absorption of nitrogen nitrogenous balance can be kept up in individuals who have become accustomed to a nitrogen-poor diet. Thus, in six individuals on a nitrogen-poor diet observed by Hirschfeld, Kumagawa, Klemperer, Sivén, and Albu, the average nitrogen absorption per kilo. of body weight amounted to 0.116 grm. per diem. These individuals were kept in nitrogenous balance. In Sivén's own case, indeed, the intake of nitrogen was only 0.08 grm. per kilo. of body weight, and nitrogen balance was kept up for a short time at least. Table I.A shows the nitrogen absorption in our cases per kilo. of body weight; it averages 0.0297 grm. over the six cases, a figure far below even the minimum absorption, when nutrition was kept up in individuals used to a nitrogen-poor diet. Table I.B shows the nitrogen balance in our six cases of rectal feeding; in all it is seen to be markedly negative. The patients were losing nitrogen from the tissues.

TABLE I.B.

Nitrogen balance :—

Observation.	N. absorbed.	N. of Urine.	Balance.
I.	1.54 grm.	3.27 grm.	−1.73
II.	1.098 „	5.5 „	−4.408
III.	1.7 „	2.4 „	−0.98
IV.	0.61 „	6.31 „	−1.69
V.	1.38 „	6.87 „	−5.49
VI.	2.22 „	8.12 „	−5.89

From our observations it may be concluded that—

1. Proteid food, even when predigested and with salt added, is very poorly absorbed in rectal feeding.

2. The albumin of eggs, as generally used in rectal feeding, is a very expensive and unsatisfactory food stuff.

3. There is no relation between the amount of proteid injected and the amount absorbed. Absorption seems to

depend more on the patient's individual capacity for absorption than on the amount of proteid food given.

Absorption of Fat.

Much of our knowledge of the absorption of fat is based on the work of Munk and Rosenstein. Their observations were made on a patient with a lymph fistula through which the absorbed chyle could be collected. The patient was kept on a fat-poor diet, then given enemata of an emulsion of 15 grm. of lipanin, with 0.4 per cent. saline solution. The lymph showed a rise in fat contents of from 0.18 to 0.45 ; that is to say, 3.7 per cent. of the oil was absorbed. In the second observation the fatty content of the hunger lymph was estimated, a cleansing injection was then given, followed by an enema of 20 grm. of lipanin in soda solution. The lymph showed a rise in fat of from 0.06 to 0.37 per cent., and after 9 hours was still 0.23 per cent. There was absorption, therefore, of $5\frac{1}{2}$ per cent. of the fat of the enema.

Kobert and Koch found that emulsified fat was slowly absorbed in small quantities, unemulsified fat was but little absorbed.

Munk and also Armschink found that fat is better absorbed the lower its melting-point.

Deucher gives observations on three patients who were kept on a fixed diet and at intervals given enemata of fat in addition. It seems to us that here, as in many observations on rectal feeding, the giving of food by the mouth is a most unsatisfactory complication of the observation. In Deucher's first observation, 6.8 per cent. fat was absorbed ; in the second, during the first period 12.6 per cent., during a second period 19.5 per cent. absorbed. In the third observation, when a small quantity of fat was given and a longer period allowed for absorption, 63.8 per cent. was absorbed.

Deucher concludes that 10 grm. of fat per diem are absorbed with difficulty even in favourable circumstances, and that the best results are got by the use of small quantities retained in the colon a long time, and with salt added to the injection.

Table II.

Table to show absorption of fats as average per diem ;—

Observation.	Fat of Enema in Grm.	Fat absorbed in Grm.	Percentage of Absorption.	Caloric Value.
I.	103.37	45.85	44	426
II.	47.44	23.87	51	222
III.	40.24	14.46	35	134
IV.	39.61	5.45	12	50
V.	9.18	- 2.55	—	- 23.71
VI.	14.35	3.47	24	32

In our observations the fats were estimated by weight after extraction with ether ; the resulting figure may, therefore, be more correctly called ether soluble bodies. The results obtained differ materially from those of Deucher. We find a definite relation between the amount of fat given and the amount absorbed. The maximum of absorption, 45 grm. in Observation I., was where the maximum 103 grm. were given in 24 hours. Considerable fat absorption was got whenever a large amount of fat was used in the enema. In Observation IV. a small amount was absorbed, 5.45 grm., equal to 13 per cent. as compared with Observation III., where with practically the same amount in the enema a considerably larger quantity was absorbed, but it is to be noted that the patient in Observation IV. was poorly nourished and exceedingly emotional and that albumin was equally badly absorbed. Observation V. is difficult to explain : the amount of fat recovered from the stool was greater than the amount injected by an average of 2.5 grm. per diem. The patient was a very well nourished, somewhat chlorotic young woman, and one can only conclude either that her fat absorption was practically nil and that the surplus amount recovered was accounted for by the digestive secretions, or that some unauthorised article of food was taken by the mouth. The latter explanation was indignantly denied by the patient and considered impossible by the nurses in charge, and is not borne out by the figures of the nitrogen and sugar metabolism.

In glancing at Table III. it is interesting to note that fat absorption seems to play an important rôle as a nitrogen saver :

the loss of tissue nitrogen for the six observations is very much larger in those where the fat absorption is poor. Taken as a whole, these observations show that emulsified fat is a very useful ingredient in rectal enemata, and is very much better absorbed than is generally considered.

TABLE III.

Table to show loss of nitrogen of tissues and loss of weight in relation to fat and sugar absorption as average per diem :—

Observation.	Nitrogen of Tissues.	Loss in Weight in Kilos.	Fat absorbed.	Sugar absorbed.	Caloric Value of Absorption.
I.	1.73	0.378	45.85	43.8	606
II.	4.4	0.245	23.87	38	378
III.	0.98	0.405	14.46	61.85	388
IV.	5.69	0.518	5.45	50.61	258
V.	5.49	0.583	– 2.55	81.1	308
VI.	5.89	0.778	3.47	36.96	184

Absorption of Sugar.

The absorption of sugar has been studied by Voit and Bauer, Schoenborn, Strauss, Leube, Plantenga, Deucher, Zehmisch, and others.

Deucher gave a patient during nineteen hours five enemata containing each 40 grm. sugar, and got an absorption of 77 per cent., that is to say, 154 grm. were absorbed. Plantenga, working with smaller quantities, got good results. Zehmisch, giving 152 grm. per rectum, got an absorption of 103 grm.

In these observations, conclusions were drawn from the amount of sugar or dextrine recovered from the stool; that is to say, a known quantity of sugar was given by the bowel, the sugar in the fæces estimated, and the difference taken as the measure of absorption. Exception has been taken to this method. It has been urged, that under the influence of micro-organisms, sugar is split up, and is absorbed or passed in the stool as the products of bacterial action, and thus much which

disappears from the bowel contents has provided no nutriment for the patient.

This view is supported by Reach. He shows that 60 grm. dextrine given by the mouth has a definite effect in raising the respiratory quotient, while the same amount given by the bowel did not definitely raise it. He considers that the amount of sugar absorbed by the bowel is very considerably behind the amount absorbed when given by the mouth. Dextrine, he finds, is better absorbed by the bowel than sugar, and does not irritate the bowel to the same extent.

We have thought it of interest to see how far sugar was broken up under the influence of the micro-organisms of the bowel, and especially the B. coli communis, the most important micro-organism of the intestine.

The observations were carried out on solutions of dextrose of known strength. To the dextrose peptone was added. A number of tubes containing 10 c.cm. of the solution were sterilised, and subsequently inoculated with B. coli communis or with bowel contents from a patient under rectal feeding. The tubes were then incubated, and the loss of sugar estimated every hour. As a rule it was found that the loss of sugar reached a maximum after four or five hours of incubation. A large number of observations were carried out. Six are given. From this table it will be seen that in all the loss of sugar was below 1 per cent., ranging from 0.49 per cent. to 0.77 per cent.

TABLE IV.

Observation.	Material used.	Incubation Time.	Loss in Sugar.
I.	Dextrose, .5 % peptone, bacillus coli comm.	4 hours	0.49 per cent.
II.	Do. do.	4 „	0.51 „
III.	Do. do.	4 „	0.56 „
IV.	Dextrose, 5 % peptone, and bowel contents	5 „	0.74 „
V.	Dextrose and bowel contents	4 „	0.73 „
VI.	Do. do.	5 „	0.77 „

Table V.

Table to show absorption of sugar as average per diem :—

Observation.	Sugar of Enema.	Sugar absorbed.	Caloric Value.
I.	47.75	43.8	179
II.	38	38	155
III.	61.85	61.85	253
IV.	57.12	50.61	207
V.	88.14	81.1	332
VI.	39.08	36.96	151

With the exception, then, of Reach's observations, which we cannot accept as conclusive, the objections to the use of sugar as not being utilised, but as being split up before absorption, are wholly theoretical. Our observations would tend to show that the amount lost by bacterial action is but small, being always below 1 per cent. In support of our view we may cite the observations of Kausch and Socin, who have demonstrated in animals that milk sugar and galactose, when given by the bowel, appear to enter the liver and are converted into glycogen.

In the clinical cases pure dextrose was used. Table V. shows the results obtained. In all the cases the absorption appeared good. In Observation III., where 61.85 grm. were given daily, all appeared to be absorbed ; in Observation V. 88 grm. were given, 81 absorbed. The absorption seems to vary in the different cases, not with the amount given, but rather with the varying capacity of the individual for absorption.

The caloric value obtained from the use of sugar ranged from 151 calories as a minimum, to 332 calories as a maximum. In none of our cases was there any bowel irritation produced by the dextrose, a disadvantage which has been urged against its use.[1] In no case did alimentary glycosuria result.

[1] The absence of bowel irritation was probably due to the use of pure dextrose. Commercial dextrose may contain impurities, such as sulphuric acid, which would induce bowel irritation.

Observation VII.

Day.	Enemata.			Excretion.				Weight of Patient in Kilogrammes.
	Nitrogen.	Fat.	Carbohydrate.	N. of Fæces.	N. of Urine.	Fat.	Carbohydrate.	
I.	2.51	15.33	130.38	1.88	12.18	3.78	20.00	57.885
II.	2.51	15.33	130.38	0.87	7.64	1.26	6.90	
III.	2.51	15.33	130.38	2.46	5.91	12.94	5.46	
IV.	2.51	15.33	130.38	3.22	5.68	17.15	5.44	
V.	2.51	15.33	130.38	3.30	6.05	19.27	5.14	
VI.	2.51	15.33	130.38	3.50	4.73	12.67	15.00	58.112
Totals	15.06	91.98	782.28	15.23	42.19	67.07	57.94	Gain 0.227 kilos.

Day.	Nitrogen absorbed.	Nitrogen of Urine.	Nitrogen from Tissues.	N. Value as Protei d of Tissues.
I.	0.63	12.18	11.55	72.18
II.	1.64	7.64	6.00	37.50
III.	0.05	5.91	5.86	36.62
IV.	−0.71	5.68	6.39	39.93
V.	−0.79	6.05	6.84	42.75
VI.	−0.99	4.73	5.72	35.75
Totals	−0.17	42.19	42.36	264.73

Caloric value of material metabolised per diem as average over period :—

Proteid.	Calories.	Fat.	Calories.	Carbohydrate.	Calories.	Total Calories.
—	—	4.15	38.595	120.72	494.95	533

[Case 7 is not included in our averages. The patient was admitted to hospital suffering from severe hæmorrhage from the stomach. During the first two days of the period of observation there was blood in the stool, and though the nitrogen of the fæces on these days is not high, we have omitted the case from our series. It is to be noted, however, that considerable quantities of sugar were absorbed, raising the caloric value of the rectal nutriment to 533 calories. The slight gain in weight is probably due to absorption of water following on the hæmorrhage.]

Table VI.

Relation between food calories used and loss in patients' weight as average per diem :—

Observation.	Food Calories.	Calories used.	Loss in Weight in Grammes.
I.	1777	645 = 35 per cent.	367
II.	939	405 = 48 „	246
III.	869	431 = 49 „	405
IV.	894	273 = 30 „	519
V.	567	344 = 60 „	584
VI.	428	240 = 56 „	892

Table VI. shows the relation between the caloric value of the food given, the caloric value of the food absorbed, and the loss in the patient's weight. The table seems to show fairly conclusively that the observation data may be considered accurate. The figures in column 1 diminish from above downwards ; in Observation I. a much larger amount of food was given than in Observation VI. Column 2, showing the calories used, diminishes from above downwards, but it is to be noted that the percentage absorption is not in proportion to the amount given : thus in Observation I., while 645 C. were absorbed (equivalent to only 35 per cent. of the food given), the wastage was large. In Observation V., while only 344 C. were absorbed, the absorption equalled 60 per cent. of the food given. The loss in the patient's weight increases from above downwards in the table. When little food was absorbed, as in Observation VI., when only 240 C. were absorbed, the loss in weight is greatest, equalling 892 grammes per diem.

The important point for the clinician is, how far can rectal alimentation be relied on as a means of nourishing a patient if gastric rest is indicated as a therapeutic measure. How far can a patient's nutrition be kept up during a period of gastric rest? The standards of food requirements which have generally been accepted are those of Voit and of Atwater. These expressed simply in their protein and energy value are :—

	Protein Grm.	Energy Value in Calories.
Voit . .	85	1860
Atwater . .	100	2700

Chittenden, however, in his recent work on "Physiological Economy in Nutrition," has established a new standard of dietary requirements. He carried out a series of experiments on himself and his colleagues, as representing professional men, and on a number of typical athletes, and found that in the athlete, as well as in the less active or professional men, health and undiminished working capacity could be maintained with an intake of proteid food far below the quantity generally consumed, and that without increasing in any measure the amount of non-nitrogenous food.

Sivén in his work shows that the organism does not suffer through a diminished supply of proteid, and can for a short time, at least, exist as well on 12 to 13 grm. of pure albumin as on a hundred, without any of its own proteid suffering; and that the individual does not lose in energy by an albumin-poor food, for energy is fed at the potential cost of the nitrogen-free food. Sivén, in his own person, was able to keep up nitrogenous balance with a nitrogen intake equalling only 0.08 grm. per kilo. body weight. Albu, working with a female vegetarian, got as nitrogen balance on a diet containing a value of 1400 calories with a nitrogen intake of only 0.15 grm. per kilo.

Paton and Dunlop, from their diet studies, are inclined to adhere to the older standards nothwithstanding Chittenden's results.

The question for the clinician is—given even a reduced standard of dietetic requirements—how far does rectal feeding meet these requirements? The caloric value of the food absorbed in our investigations varied from 645 calories as a maximum to 240 calories as a minimum, giving an average of 389 calories. Thus we get from rectal feeding only about a quarter of the nourishment required to maintain equilibrium, even if a reduced standard of nutritive requirements be accepted. In considering these cases it must be remembered that they are all cases where rectal feeding was well borne, the conditions were in fact favourable to food absorption. If the rectal feeding was not well borne, if diarrhœa or vomiting was at all a prominent symptom, or if the patient's nutrition was obviously suffering under the rectal feeding, other methods of treatment were adopted and the observation was not included in the series.

The caloric value obtained in our cases is much below

that claimed by Leube with meat and pancreas enemata. Riegel speaks well of Leube's enema; in our hands it proved unsatisfactory. It was difficult to administer, the pancreatic value of fresh pancreas was found very variable, and intestinal putrefaction was such a marked feature after its use as to cause a pronounced toxæmia. Both the patient who received the enema, and the nurse who administered the daily cleansing enema, suffered severely.

From our series of cases we must conclude that rectal feeding, even under the most favourable circumstances, is sub-nutrition of a most pronounced character. Given a patient in poor condition, it cannot be relied on to produce any material improvement in nutrition. This seems an important point to emphasise. The surgeon frequently wishes to have a patient suffering from œsophageal or pyloric obstruction brought into a condition of improved nutrition before operation. It cannot be too strongly emphasised that rectal feeding cannot be relied on to produce this desired result. It will at once be urged that patients may gain weight under rectal feeding. In considering this the absorption of water from the intestine must be remembered. After severe hæmorrhage or persistent vomiting, as in pyloric obstruction or gastric ulcer, the tissues are suffering markedly from a deficiency of water; on placing the patient on rectal feeding, water is absorbed and there may be a gain in weight. This is well seen in Observation VII., where rectal feeding was begun after a very severe hæmorrhage from the stomach. Though the observation shows a marked negative nitrogen balance and an absorption of 500 calories, there was a gain in weight after six days' rectal feeding.

If the physician desires to make use of rectal feeding, it seems from our observations that the best results will be got from the carbohydrates and fat. The absorption of proteid food from the bowel is so small as to make it of little value as a food stuff.

Folin has recently enunciated a theory of proteid metabolism which would place proteid on a much lower scale as a food than is at present admitted. He regards proteids, in fact, as of secondary importance, inasmuch as their nitrogen is split off and eliminated, leaving the carbon and hydrogen containing part to be oxidised, and thus to yield energy as it is yielded by the carbohydrates and fats. Folin's views

have not so far met with general acceptance (Paton), but even without accepting Folin's views, we may evidently with advantage abandon much of the nitrogenous material which is at present used in rectal alimentation.

There remain, then, the carbohydrates and fats. In regard to carbohydrates, in our observations pure dextrose was used and gave most excellent results. In all cases it was well absorbed and produced no ill effects, such as bowel irritation or alimentary glycosuria. In Observation VII., the caloric value of the food-stuff absorbed was raised to 500 calories by increasing the quantity of sugar. During the whole period of observation the patient was comfortable, and did not suffer from hunger. The disadvantage of pure dextrose is that it is somewhat expensive.

The disadvantage of fat in rectal feeding seems to be the wide difference of absorptive power of different individuals, as is very well seen in our observations. When well absorbed it should prove most valuable, but the absorption cannot be foreseen in any given case. It is undoubtedly best given in the natural state, as in the yolk of egg. If prepared fat is used, it should be fine emulsion of a fat of low melting-point. Pure olive oil may be used, a small quantity being saponified and used to emulsify the whole.

For practical purposes a good nutrient enema consists of—

The yolks of two eggs.
30 grm. pure dextrose.
.5 grm. common salt.
Pancreatised milk to 300 c.cm.

The approximate caloric value of such an enema equals 300 calories. Given every six hours the total nourishment injected would equal 1200 calories. Absorption under favourable circumstances might equal 500 calories, but most probably would be much less.

In rectal feeding the method of administration and the size of the enema are both important points. Nutrient enemata should never be administered with a syringe. To place a syringe in the hands of a nurse is to court failure. Substances rapidly injected into the bowel will as promptly be returned. The enema should be very slowly syphoned in

by means of a soft rubber catheter and a small-sized filter funnel. Retention of the nutrient material is not then a matter of difficulty. If there be bowel irritability, a small dose of morphia may be added to the injection. The daily cleansing of the bowel with a saline injection is, of course, an absolute necessity.

The amount of the nutrient injection commonly used in this school is from 4 to 6 oz. (112 to 168 c.c.). This is possibly too small an amount. If the enema be given slowly, 8 to 10 oz. (250 to 300 c.c.) can be retained, and the patient absorbs at least sufficient water and does not suffer from thirst, a prominent difficulty when small injections are used. Larger injections do not require frequent repetition, a distinct advantage where gastric rest is desired, for it must be remembered that the injection of nutrient material into the lower bowel excites gastric secretion, and may thus prevent the healing of a gastric ulcer, and account for the gastric pain so frequently complained of in gastric cases under rectal feeding. This gastric secretion, though long recognised clinically, has been clearly demonstrated recently by Umber in a patient on whom gastrotomy had been performed. After a rectal injection there was a free gastric secretion, with a total acidity of 30, free hydrochloric acid equalling 20.

It is the popular belief that substances injected into the lower bowel are absorbed below the ileo-cæcal valve, but this does not seem always to be the case. Insoluble particles injected into the rectum may, under favourable conditions, reach the stomach. Grützner has shown that if starch emulsion be injected into the rectum along with normal saline solution, the starch granules can be washed out of the stomach four to six hours afterwards. The passage of the nutrient material beyond the ileo-cæcal valve may account for the good results got in some cases of rectal alimentation, and is an argument in favour of increasing the bulk of nutrient enemata when the patient can tolerate the larger injection. That the ileo-cæcal valve will admit of the passage of fluid in every case cannot, however, be expected ; in fact, we have frequently failed to find insoluble particles, such as charcoal, in the stomach washings in cases where charcoal was added to the nutrient enema.

The conclusion to which this study of the metabolism leads

seems to be that rectal alimentation has a much more limited field of usefulness in clinical medicine than is at present accepted. In the treatment of acute gastric diseases where it is so commonly employed, it interferes with complete gastric rest by inducing gastric secretion, and does not sufficiently nourish the patient.

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2. ON BIER'S TREATMENT OF ACUTE INFLAMMATION BY PASSIVE CONGESTION

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AS the outcome of Lister's antiseptic method, surgeons everywhere now recognise that acute inflammation and suppuration in operation wounds, made through unbroken skin, are complications which need not arise, and for which they and their assistants are responsible. But although Lister's immortal method has prevented the onset of an incalculable amount of suppurative disease, with all that is thereby entailed, the method has done, and can do, comparatively little to cure suppurative disease when it is actually in progress. So long as the pyogenic organisms are outside the body they can be dealt with effectively by many well-known means. When, however, they have become lodged in the body, they are safe from the weapons which could have destroyed them when outside. This is partly because they are then protected from the attack of antiseptic lotions by the tissues and fluids in which they lurk, partly because the tissues are generally more liable to be hurt by the antiseptic measures than the organisms are. Thus it is that the surgeon is still at a great disadvantage when he has to deal with such cases as a whitlow extending into the tendon sheaths, an acute suppurative osteo-myelitis, a large boil or carbuncle, suppuration in a joint from accidental wound or otherwise, or with any form of suppurating wound, whether contaminated by traumatism, or as the result of failure of his own antiseptic precautions. In all such cases the agents which most effectively attack the invading organisms are the cells and fluids of the invaded part, and that surgeon succeeds best who best aids and abets these weapons of nature, whether with or without the addition of antiseptic agents technically so called.

But while the wisdom of aiding natural methods of cure for suppurative inflammation is well recognised, there has long been much uncertainty as to what these natural agents really are, and as to the way in which they are brought into play. Recent discoveries have indeed thrown some light on this subject, but the knowledge we now have is still far from being definite and clear. Our fathers in the profession considered inflammation hurtful, a process which must be combated, as the word antiphlogistic clearly shows. Bier, on the other hand, looks upon

inflammation as a curative agent which ought to be encouraged and utilised rather than opposed and thwarted. Whether Bier's view or the old one is the more correct we need not stop to discuss just now. This at least is certain that, based upon his theory of inflammation, Bier has devised a practical method of treating suppurative diseases which is astonishingly successful, and no less simple than effective. By the use of a piece of elastic bandage properly applied—as I hope to show—the surgeon is enabled to do nearly as much to cure septic inflammation of the extremities as antiseptic and aseptic methods have done to prevent them. Prevention of course is, and will always remain, better than cure; but, in many kinds of suppurative disease, prevention is impossible, and when this is so a method of treatment which cuts these diseases short and robs them of their terrors cannot fail to be of inestimable benefit to mankind. For this reason I am glad of the opportunity of bringing under the notice of the Medico-Chirurgical Society a method which is comparatively unknown, but which I am certain will before long be recognised as one of the standard methods of treatment in the hands of the profession.

As the time is limited I shall not attempt to give an historical account of what led Bier to adopt this method of treatment. It is sufficient for the present purpose to say that he has employed passive congestion for acute suppurative inflammation since 1893, although more extensively in the latter part of that time.

The method he adopts as a general rule is as follows:— Suppose the case to be one of acute whitlow, extending along a tendon sheath to the wrist, and producing redness and swelling of the hand. A piece of Martin's rubber bandage would be bound round the upper arm. If the foot were affected the bandage would be applied to the thigh, *i.e.* always a considerable distance above the seat of mischief. The bandage is put on sufficiently tight to hinder definitely the venous return, while it only slightly diminishes the arterial flow. The pressure in acute cases is very light. Any accessible pulse below the bandage should be easily felt, and the limb should be warm. One great test of the proper degree of tightness is the relief of pain. Sometimes a good deal of patience is required before the exact degree of tightness is attained which diminishes pain. When this has been found

the rapid relief of pain is very striking, and generally astonishes the patient's friends as much as it gratifies the patient. In a few cases, however, and these chiefly of gonorrhœal origin, pain may be increased at first, although it afterwards diminishes. Another test of the proper application of the bandage is the objective effect upon the limb. After a few minutes the whole limb begins to swell, and the "fiery red" colour, formerly limited, say, to the hand and wrist, will now extend right up to the elastic bandage, while the skin will also feel warmer. When the œdema is well established, serum escapes freely from any wound which may exist, and an appropriate amount of loose absorbent dressing will be required to soak it up. There must, however, be nothing tight round the limb below the bandage. Severe cases should be treated in bed. In bad cases the passive congestion is kept up for 22 out of the 24 hours, and this may be diminished by degrees to from 8-10 hours. The following points, however, have also to be attended to :—

The position of the bandage should be changed after about 10 hours in one place. The bandage over an œdematous limb may have to be tightened as the œdema is dispersed.

In parts of the body, such as the shoulder, where there is no room to vary the position of the bandage, Bier recommends that congestion should not be kept up for longer than 8-10 hours, and that the part compressed by the bandage should be well washed with spirit at each interval.

In the intervals of relaxation the limb should be raised so as to favour absorption of the effused lymph.

The progress of the case can be gauged by the patient's general condition and by the local appearances and symptoms. As a general rule the patient is not only soon eased of pain, but feels brighter and better. The pulse improves and the temperature drops. This reduction in the temperature occurs very quickly when the cause of the raised temperature is merely the absorption of poison from the inflamed area. A rise of temperature may then be observed during the intervals in which the bandage is relaxed. This of course is due to the setting free again of poison retained along with the serum. In proportion, however, as the treatment is successful in combating the suppurative inflammation, such rises of temperature diminish since there is less poison generated in the local

micro-organismal factory. On the other hand, when the cause of elevated temperature is septicæmia or pyæmia, passive congestion can only influence the temperature indirectly by improving the condition of the focus from which the circulating blood has been contaminated; and such influence may, for a time at least, be inappreciable.

At the affected place, the more acute the inflammation, the more easy it is to cause œdema and diffuse redness by the elastic compression. As the inflamed area improves by the use of the bandage, it responds less and less to constriction on the limb, until the reaction is almost as little as it would be in health. The surgeon, however, does not require to keep up the passive congestion as long as this. After convalescence has fairly set in, progress seems to go on faster without artificial aid. This of course is true of many other remedies besides the one we are now speaking of. Any pus which may form should be let out, but the incision or incisions need not be so extensive as would have been necessary without passive congestion. Free incisions are only required when the circulation of the part is seriously impaired. Should blisters appear, either the bandage is too tight or suppuration has occurred which must be relieved.

Bier insists upon great care and watchfulness, especially in the first day or two. If the bandage is too slack, it does no good; if too tight, it will do harm. Medical men who have not yet gained confidence in the method are advised to begin with the milder cases, such as (1) all quite recent acute inflammations; (2) subacute and less severe inflammatory affections, whether recent or not; (3) gonorrhœal, pyæmic, and acutely suppurating joints, before the tendon sheaths and bones have become involved. After serious complications have arisen the cases are more difficult to treat, and the results, which are not likely to be quite satisfactory at the best, may be put down to the passive congestion method, and not to the damage done before it was begun. Still less should anyone who is not familiar with the method and convinced of its value employ it in dangerous cases. In spite of its use, amputation is often necessary in such cases, and death may follow. An undeserved discredit might then be thrown upon the method, which would prevent its being used in cases where its value would be unmistakable. In general practice, Bier advises that

this treatment should only be employed by medical men who have had experience in surgery, and by them only when they are able to keep the patients under close observation.

In contrasting the use of passive congestion for tuberculosis and for acute inflammation, Bier points out that in tuberculosis the congestion is now only kept up for one or two hours daily, and that cyanosis but no œdema results; while in acute inflammation the congestion is prolonged for the greater part of the 24 hours, and the limbs become fiery red in colour and very œdematous.

In order to give an adequate idea of the scope and value of Bier's method, I shall now quote a few typical cases published by him. Only one case—and that where possible a short one—has been selected out of some of Bier's groups of cases.

Suppression of commencing Suppuration by the Use of Passive Congestion.

On the 4th of February 1904, an almost inoperable scirrhus of the breast was removed from a woman aged 60. The wound suppurated freely and for a long time. On the 8th of April a pyæmic metastasis set in in the left wrist, accompanied by a rigor and rise of temperature to 39.4° C. (103° F.). The joint was very red, swollen, and tender on the surface. Passive congestion was applied at once and maintained for 20 hours each day. The pain subsided rapidly, and by the 11th of April the affection of the joint had completely disappeared; temperature was normal and passive congestion was stopped.

Transformation of Acute into Cold Abscess.

This Bier states to be a rare occurrence, but as a well authenticated instance of such transformation is of considerable pathological interest, I shall quote the following case:—

A boy of 7 was admitted to the Klinik on the 28th of July 1904 on account of a sharp and very painful acute inflammation of the lower part of the right thigh. There was redness and swelling on the whole of the outer side, and at the centre there was distinct fluctuation. A diagnostic puncture showed that this contained thick pus with staphylococci. The abscess was purposely not opened, but simple passive congestion was begun. By the 30th of July the abscess had become cold. All appearance of inflammation had disappeared, and the previously elevated temperature had sunk to the normal. The

abscess was, however, of the same size as before, and had caused thinning of the overlying skin. On the 5th of August a thorn 1 cm. long escaped, and through the small opening thus made the pus was pressed out without further opening in the skin. On the 9th of August the boy was dismissed with his thigh quite healed.

In another similar case which Bier narrates, the pyogenic organism was a streptococcus.

Disappearance of Abscesses under Passive Congestion.

A boy of 12 was admitted on the 23rd of July 1904 with a severe carbuncle-like inflammation of the back of the head and neck. The skin over the occipital bone on both sides and down to the middle of the neck was densely infiltrated, and the neighbouring parts were much swollen from œdema, and very tender. In the centre of the hard swelling there was a soft portion about the size of a walnut. This was found by diagnostic puncture to contain thick pus with staphylococci. The patient kept his head rigid and bent backwards, temperature in the axilla only slightly raised, *i.e.* 37.9° C. (100° F.). An elastic bandage was applied to the neck and worn during the day. By the 26th of July the head was freely movable and the affected part free from pain. The acute (*heiss*) inflammation had become cold. A puncture now made into the soft spot revealed only clear serum. The passive congestion was continued, and when the patient was dismissed on the 2nd of August all traces of the inflammation, including the abscess, had disappeared.

In commenting on this case, Bier wishes it to be understood that he has only allowed abscesses to be absorbed under passive congestion in a few cases where the symptoms were otherwise favourable. By so doing he has been able to demonstrate the powerfully curative action of the treatment. In ordinary cases his rule is to open an abscess as soon as the presence of pus has been recognised.

From the pathological standpoint, the absorption of pus, after an acute abscess has been transformed by passive congestion into a cold one, is of much interest. Bier believes that occasionally the same process takes place spontaneously in some cases of appendicitis and in some cases of osteo-myelitis. Moreover, in children, as is well known, non-tuberculous empyemas may become absorbed after one or two tappings,

and in general surgical practice it is not uncommon to meet with cold abscesses which had begun by being acute, and which, when opened, contained only pyogenic organisms. These facts, together with Bier's evidence as to what takes place when the natural processes are stimulated by passive congestion, enable us to realise that the spontaneous absorption of pus, due to pyogenic organisms, probably takes place more frequently than we have generally supposed, not merely in the abdominal cavity, but also in other parts of the body.

Behaviour of Suppurating Surfaces under Passive Congestion.

As a general rule the pus becomes at first increased in quantity and thinner by the addition of serum; sometimes it is more plentiful, and yet thick at first, although it soon becomes thin. In five cases Bier notes the development of a bad smell and gas, but otherwise these cases went on well. In a very short time the discharge of pus ceases, sloughing tissue is thrown off, and what is more important, portions of tissue recover which apparently would have died under the ordinary methods of treatment. Moreover, suppuration becomes localised and limited, and the incisions necessary to let it out are less extensive and mutilating than they would otherwise be.

Treatment of Acutely and Subacutely Inflamed Joints by Passive Congestion.

Bier narrates several cases of gonorrhœal joint inflammation treated with great benefit by his method. I shall not quote any of them, however, because one of the cases which I have had the opportunity of testing the method upon was a gonorrhœal one, and our time does not admit of more than one case of each kind. In joints affected acutely or subacutely by rheumatism, great relief to pain is given by passive congestion, but in these cases the affection is fleeting in any case. In all such cases, however, Bier recommends the treatment, and he has had very satisfactory results in two severe cases of joint inflammation in puerperal fever.

Treatment of Acute Suppuration in the Larger Joints.

Besides the application of the passive congestion to these cases, Bier mentions certain other details of treatment which he has arrived at as the result of experience.

From joints in which there is a fistulous opening discharging pus, the pus is made to flow every day by passive movement; but existing openings, however small, are not enlarged and the joints are not drained. An unopened joint containing pus is punctured with a fine needle merely for diagnostic purposes, but is neither opened nor drained; no splints are applied. Reliance is placed entirely on passive congestion. Should the knee be affected the patient is kept in bed; if it be the elbow, the affected arm is supported in a sling when the patient can go about; if he is in bed, the arm is allowed to lie in any position that is comfortable. Discharge is taken up by any kind of loose absorbent dressing. Active and gentle passive movements of the joint are begun as soon as the anæsthetic effects of the passive congestion permit—generally in a few hours. These movements are repeated once a day at first, more frequently afterwards. The combination of early movements with passive congestion has brought about a complete restoration of function, without the slightest restriction in even the most severe cases of suppuration in joints.

The following is a typical case of a joint which had a small open wound:—

A stone-breaker aged 18 was stabbed in the left elbow during a scuffle on the 25th of May 1904. The joint was infected and the patient was admitted on the 11th of June. The left elbow was very much swollen, and was red and œdematous. It was held at nearly a right angle and only about 15° of active and passive movement was possible, and that with pain. Over the radio-humeral joint there was a round opening from which projected a soft mass of granulation tissue about the size of a hazel-nut. On passive movement a quantity of pus flowed from this opening. The pain was so great that it prevented sleeping. Temperature was normal. Passive congestion was begun at once. On the 2nd of July the fistula was closed and the swelling of the joint had subsided. Passive congestion was stopped and the joint treated by passive movement. On the 12th of July he was dismissed with all the movements of the joint free, except a slight inability to extend completely. On

the 9th of August the patient reported himself with a completely normal joint. He had resumed his hard work as a stone-breaker without any inconvenience.

In the following case there was no opening into the joint :—

A locksmith, aged 20, was suddenly seized on the 4th of July 1904 with an acute inflammation of the right ankle-joint. The affection, after flitting from one joint to another, finally settled with great severity in the right knee, 6 days before the patient's admission to Klinik on the 27th of July. The right knee-joint measured $6\frac{3}{4}$ cm. more than the left, and the whole thigh was very œdematous and numerous veins were visible on the surface. The joint, which had been kept on a splint, was extended and exceedingly tender. Anything which shook the bed caused the patient to cry out with pain. No attempt at movement was possible. A diagnostic puncture with a hypodermic syringe revealed pus which contained staphylococci, as shown both by culture and the microscope. Passive congestion was begun at once, and some trouble was necessary before the right amount of congestion could be reached. After this had been attained, improvement went on so rapidly that in four days the pain had almost completely disappeared. After 12 days of the application of passive congestion, the previously elevated temperature had sunk to normal, although there had been another rigor on the 3rd of August. On the 10th of August the patient was able to flex his knee to a right angle. There was still considerable effusion in the joint ; but when a specimen was withdrawn with the hypodermic needle it was found to be of a turbid serous character. After the 10th of August the passive congestion was applied for only 2 hours a day. The patient got up from the 15th of August onwards, and although there was a slight effusion in the joint it soon disappeared. Afterwards he acted as a ward attendant in the Klinik. The suppuration in the knee-joint left no trace behind it.

Treatment of Suppurative Inflammation of Tendon Sheaths.

There are probably no cases in which the passive congestion treatment gives better results than those of whitlow, if they are only seen early enough. On the other hand, if suppuration be allowed to burrow unchecked in the sheaths of the tendons for even a day or two, the vitality of the tendons may be irremediably destroyed. Hence passive congestion, unless applied at once, can only be expected to palliate such cases, not to cure

them completely. Even such palliation, however, will often be a great gain, as compared with what might have been the result otherwise.

Bier recommends that pus should be let out as soon as its presence is recognised, by small incisions—numerous if necessary—but without either drainage tubes or packing.

The following is a good example of a severe tendon sheath inflammation nipped in the bud :—

A butcher, aged 43, accidentally tore the skin of the little finger of the left hand on the 2nd of November 1899, making an oblique wound about 2 mm. broad. He took no notice of the injury, and worked on till the 5th of November. The wound then suddenly inflamed, and patient's medical attendant incised the finger three times. As the condition became much worse, the patient was admitted to the Greifswald Klinik on the 11th of November. On the flexor aspect of the second phalanx of the little finger there was an oblique wound in which the tendon was exposed. The whole course of the flexor tendons of the little finger was exceedingly tender to touch as far as the wrist. On stroking along the line of the affected tendons from the wrist towards the wound a dirty watery fluid escaped. The skin over the ulnar portion of the wrist and for 3 cm. above it was red and tender. The joint about the first and second phalanges of the little finger was open. There was extensive lymphangitis of the forearm and swelling of the supra-trochlear lymphatic gland. The patient felt ill and was in great pain. Temperature, 39° C. (102.1° F.). Passive congestion was applied at 11 A.M. on the 11th of November, and considerable trouble was necessary before it could be properly adjusted. When this was attained, the pain rapidly subsided, and the finger, which previously had been very painful and immovable, could be moved voluntarily. At 4 P.M. the elastic bandage was removed, and the pain very soon returned. At 6.45 P.M. there was a rigor. Passive congestion was reapplied ; again there was difficulty in attaining the proper degree of congestion, but with the same satisfactory result as before when it was properly applied. At 8.30 P.M. temperature was 38° C. (100.4° F.), and at 10 P.M. it was 38.7° C. (101.6° F.). At 10 P.M. the congestion was removed for a short time, and then reapplied until 4 A.M. When the œdema had partially subsided, it was observed that the swelling and redness of the wrist had disappeared as well as the lymphangitis, and tenderness remained only in the neighbourhood of the flexor tendons of the wrist. On the 12th of November, passive congestion was induced from 9 A.M. to 1 P.M., from 5 to 8.30 P.M. ; and from 10 P.M. to 10 A.M. on the 13th November. By that time the affection was practically well, the swelling had

gone down, although some tenderness still remained in the old place. The passive congestion was maintained with long intermissions until the 15th November. On the 12th of November the temperature did not rise above 38.6° C. (101.5° F.), on the 14th not above 37.7° C. (99.8° F.), and from the 15th was normal. A few superficial portions of dead tendon were discharged from the wound; afterwards it healed rapidly. The joint, which had suppurated, remained somewhat stiff and creaked on movement, otherwise the functions of the hand were completely restored.

Out of twenty-two cases of suppurative inflammation of tendon sheaths treated by passive congestion, 14 recovered with sound tendons. Of the 8 cases in which there was sloughing of tendons, the mischief had been going on for more than a month in one case, for 14 days in two cases, for 12 days in one case, for 10 days in one case, for 7 days in one case, for 2½ days in one case, while in one case there was no exact history of the onset, as the patient suffered from syringomyelia.

Treatment of Acute Osteo-Myelitis.

The following is an example of how the disease may be cut short when the treatment is begun early :—

A boy, aged 13, was taken ill on the 28th of October, after a fall, and had a rigor and much pain with severe inflammation in the left leg. On the 30th October the boy was admitted to the Klinik. Temperature 38.8° C. (102° F.), rising in the evening to 39.3° C. (103° F.). He was thin and pale, and the left leg, which was very much swollen and measured 4 cm. more than the right, was reddened and very painful. There was great tenderness, especially at the upper part of the tibia. Passive congestion was begun, and at the end of twenty-four hours all subjective symptoms had disappeared. After four days all objective symptoms had also disappeared. Temperature normal. On the 4th of November the boy was dismissed quite well.

As an example of the value of the treatment in a more severe case we may take the following :—

A boy of 9 was taken ill a week before his admission with severe inflammation of the right leg and knee-joint. On the 22nd of December 1903 he was admitted very ill and with high temperature. The right leg was very much inflamed. At the upper end of the tibia there was an abscess which had been laid open by an incision 6 cm. long, which had exposed the bone. The knee-joint was much swollen and contained pus with

staphylococci, when punctured with a hypodermic needle on the 29th of December. On the same day passive congestion was begun, and in a very short time all trouble in the knee subsided. On the 9th of January 1904 an abscess was opened on the outer side of the knee by an incision 3 cm. long. On the 10th, a minute sequestrum was removed from the front of the tibia. As all symptoms of inflammation and fever had subsided, the passive congestion was not applied after the 31st January. The knee was free from fluid and movable, but two fistulous openings remained over the tibia. These were dealt with under anæsthesia on the 2nd of March, and one was found to lead to the surface of the periosteum, and the other to pass by a track lined by granulations through the head of the tibia into the knee-joint. When this sinus was scraped out some loose flakes of cartilage were removed, along with granulation tissue. Movements of the joint caused clear synovial fluid to escape. Passive congestion was induced again, and by the 25th of April the operation wound was completely cicatrised. On the 20th of May the child was discharged. There was no effusion in the knee and the range of joint movements was complete, both active and passive. Some swelling of the capsule itself, however, remained, and there was a considerable degree of lateral movement, for which a supporting apparatus was used.

In all, Bier has treated 14 cases of acute suppurative osteomyelitis with passive congestion. Of these, 6 cases healed without necrosis, 5 with very little necrosis, 2 had extensive necrosis, and 1 patient died of pyæmia, although his death had nothing to do with the treatment. This list does not include cases sent to him early and cured by passive congestion.

Apart from the fact that nearly the half of these cases were cured without necrosis, Bier is pleased to be able to record that the sequestra which did form were of small size.

Various Applications of the Method.

The constricting bandage is not only applied to the limbs, but also to the head and neck, and of this an illustration has been given. Thus even acute suppurative inflammation of the middle ear can be brought within the influence of passive congestion.

Bier has found the method of great service in all forms of acute inflammation and suppuration, besides the more serious affections which have been specially mentioned, *i.e.* boils, carbuncles, lymphangitis, erysipelas, acute eczema with itching.

What is also interesting is that the benefit is seen in inflammation which is not due to micro-organisms, but to such chemical poisons as that inoculated by a gnat bite, or derived from a gouty diathesis.

When the inflamed area is not on a part to which passive congestion can be applied, Bier uses suction to produce the desired result. His assistant, Dr Klapp, has, at his suggestion, devised various forms of suction apparatus suitable for different parts of the body.

Confirmation of Bier's Results in the Royal Infirmary.

Some may wonder why so much of this paper has been taken up with cases published by Professor Bier. If this is so, the answer is easy. No one has had so much experience of the method as he has. Only by giving details of the cases and of the results of the treatment can an adequate conception of the value of the method be obtained. Moreover, everyone who has read Professor Bier's writings, and especially if they have also had the pleasure of making his acquaintance, as many of us did last summer, will be satisfied of the absolutely reliable character of all that he records.

At the same time it is important to know if other surgeons have been able to obtain, with his method, results like his. My own experience of the method has been on the whole not only satisfactory, but surprisingly so.

The first case in which passive congestion was adequately applied in Wards 17, 18 for acute suppurative inflammation, was one of suppuration extending from the little finger into the forearm. The patient was admitted to the Royal Infirmary on the 17th of July 1905, and Professor Bier very kindly came over from the Quater-centenary garden party at Heriot's Hospital on the 21st to show me how to apply the bandage.

John Oddie, aged 36, injured his hand on the 6th of July. A small piece of skin was abraded over the first joint of the little finger of the right hand. He paid no attention to it at first. On the 7th the little finger became painful. On the 8th the whole finger became swollen and more painful and the patient came to the out-patient department of the Royal Infirmary. The wound was swabbed with pure carbolic acid and an ichthyol dressing applied to the fourth and fifth fingers. On the 10th, when the hand was being dressed, the skin and nail of the little finger

peeled off. An incision was made into the skin over the distal part of the fifth metacarpal bone, and ichthyol reapplied. Swelling and redness spread up the forearm until on the 17th, when the patient was admitted, the little finger was gangrenous and there was suppuration on the radial side of the forearm at the wrist. This was incised, and the arm put into an ichthyol bath. On the 20th an incision was made into the ulnar side of the forearm on account of the suppuration there. On the 21st the arm was put into a corrosive sublimate bath. In the afternoon, arm was taken from the bath and passive congestion applied by Professor Bier about 4 P.M. Patient was comfortable all night. 22nd, arm looking very much better. Skin wrinkled and less red. Temperature and pulse both about 98. Bandage removed 7 to 10 A.M. and reapplied from 1 P.M. to midnight. 23rd, arm improving wonderfully. Temperature normal. Congestion from 2 A.M. to 8 P.M., reapplied at midnight. 28th, wounds now granulating. 3rd August, discharged to Convalescent—little finger separating slowly. The little finger was eventually removed. The hand otherwise recovered with free movement and complete usefulness.

Although in this case improvement had begun before passive congestion was applied, the ultimate recovery at that time was doubtful. From the time when the passive congestion was applied, the progress was so decided that no further anxiety was felt.

Cellulitis of Hand and Forearm.

Robert R., æt. 40, cowman from Bathgate, was admitted to Ward 18, Royal Infirmary, on the 8th of November 1905, on account of pain and swelling in the right hand and forearm. About a fortnight before admission he had felt some pain in the thumb, which he thought was caused by the prick of a thistle, but he paid very little attention to it until the 5th of November, when it became much worse, and he was unable to sleep for the pain. An incision was made into a swelling on the ball of the thumb and various dressings were applied, but without giving relief. Hot poultices had eased him more than anything else, and by their means he had slept two hours on the night of the 7th, *i.e.* before he came in. On the 8th, when he was admitted, he was haggard and anxious looking; the hand was dark red in colour and much swollen, and the redness and swelling extended irregularly up the forearm to the elbow. He was in great pain and felt very miserable. At 2 P.M. an elastic bandage was applied to the middle of the upper arm, and at 7 P.M. the following note was made. The patient feels "a guid bit easier since it has been pitten on." The pain running up the inner

side of the arm into the arm-pit has completely vanished. There is no throbbing in the hand or stounding as before. The fore-finger can be moved a little at the metacarpal joint. (He volunteered this information.) The improvement took place about three hours after the bandage was applied, and since then he has had a couple of hours' sleep and seems refreshed and his expression is not so anxious. There is great œdema of the hand and forearm below the bandage.

9th. The patient said that he "felt like a new man." From this time there was no further anxiety as to the spread of the mischief. A slough about the size of a hazelnut separated from the pulp of the thumb on the 17th November, and an abscess was opened over the palmar aspect of the thumb on the 24th of November. On the 12th of December the patient was discharged with the wounds nearly healed, although there was still some stiffness of the wrist and fingers.

Case of Subacute Inflammation and Ulceration of the Foot.

John D., æt. 44, had suffered for about 3 weeks from an ulcer on the foot near the ball of the great toe, accompanied by inflammation of the skin of the greater part of the foot. There was an occasional shooting pain, but what the patient most complained of was numbness on the outer side of the foot, extending into the sole. He had been troubled with cramp in both legs. Fearing that there might be some interference with the blood supply, and that there was a risk of moist gangrene supervening, I drained an abscess on the dorsum of the foot and used various antiseptic applications, without inducing passive congestion. As there was little or no improvement, however, I cautiously used the elastic bandage, giving instructions to the House Surgeon to remove the bandage if it seemed to do any harm. Instead of doing harm, however, it did great good, the redness and swelling soon subsided and the ulcer began to heal, and on the 6th of November he was discharged cured.

Case of Gonorrhœal "Rheumatism" of the Knee.

Mrs X., aged 38, was suddenly seized with very severe pain in the left knee about the beginning of October. The pain came on without apparent cause while she was sitting in a friend's house. She had to go home in a car and was carried upstairs on reaching her own house. There was very little rise of temperature or pulse, but the pain was extreme from the first, until I saw her four weeks later. I found that her husband had acquired gonorrhœa from another woman in July, and had

had connection with his wife while the discharge was still present. Mrs X. had noticed a vaginal discharge but had paid no heed to it, thinking it was only leucorrhœal. When I first saw the patient she was exhausted from want of sleep and continued pain, and had a careworn, anxious look. She could scarcely bear one to touch the knee, and was in great distress if the bed were shaken or if one walked with a heavy step across the floor. The knee was swollen, but not distended with fluid, and its temperature was not raised. A piece of elastic bandage was applied above the knee with sufficient firmness to produce slight venous obstruction. I had fortunately succeeded in getting the proper degree of passive congestion at the first trial, and in fifteen minutes there was a distinct feeling of relief. From that time she steadily improved, and she is now walking about, gaining strength in the joint, although there is still a good deal of stiffness. I have been afraid of awakening the disease again by forcible movement, so we are trusting to active and passive movements, massage, and hot fomentations. Her own medical attendant has kindly sent me the following account of Mrs X.'s case, which he allows me to publish: "For nearly a month I tried everything I knew to relieve the pain—extension, elevation, fomentations, plain and medicated, liniments, iodine, etc. Internally, salicylates, potassium bromide, potassium iodide, etc., with, I may say, no alleviation of the pain at all. Night after night I was sent for, and in the long-run had to give morphia, and even this required large doses. I have never met with such an obstinate case, and she was being worn out from want of sleep and constant pain.

"Within a few hours—actually in the first hour—after putting on the elastic bandage, she felt eased, and the pain very soon left her altogether, and has not returned. It acted like a charm and gave relief at once, as well to the patient as to me."

Case of Gummata of Tibia. Considerable Wearing Pain.

Christina P., aged 13, was admitted to Ward 17, Royal Infirmary, on the 21st of November 1905, suffering from pain in the left leg below the knee and two ulcers over the tibia. In March she had been kicked on the shin and the leg had become swollen and painful and an abscess had formed. In May this had been opened and the bone scraped. A few weeks after the wound had healed the leg began to swell again, and the wound re-opened and had discharged ever since. Latterly the pain in the leg had been very severe, and made it necessary for her to have something done to relieve it. She had a depressed bridge of the nose, opacity of the cornea from previous keratitis, and syphilitic

retinitis. There seemed little doubt that the cause of all these conditions was inherited syphilis, although her father was not aware of having had syphilis. We tried the internal administration of iodide of potassium. Not only was no relief given to the affected leg, but a painful periosteal node developed meanwhile on the other tibia, and the child was kept awake at night by the pain. We now employed the elastic bandage to induce passive congestion in both legs. From that time the pain subsided and the ulcers healed. In about a week the patient was sent home, with directions to continue the elastic bandage at night and report herself if the pain returned. Up to the middle of February we had not heard of her again.

Besides these cases treated in the wards, we have used the method in a number of minor cases, some of early stage of acute inflammation where the pain was relieved and the inflammation checked, some where chronic inflammation was induced to heal.

I have had no cases where any harm was done by the use of passive congestion, but in one or two there has been no benefit. In one case of septicæmia associated with a form of erysipelas of the arm, the disease was not appreciably affected by the use of passive congestion. In another case, a very indolent form of diffuse inflammation of the side of the face and neck, accompanied by considerable pain passive congestion increased the pain even when very lightly applied. In this case hot poultices relieved the pain and brought about some concentration of the swelling, but the patient is still under treatment, and has not yet either developed suppuration or given any definite sign of resolution.

When we try to answer the question, "How does passive congestion act in acute inflammation?" we find ourselves confronted with a number of interesting problems. So far as the relief of pain is concerned, the effect is so rapidly produced that a mechanical action seems highly probable. Professor Bier suggests that the anæsthetic influence of œdema on the fine nerves is brought into play, and this view seems to answer the requirements of the case. The more active the inflammation, the easier is œdema produced; the more chronic the inflammation, the more difficult is it to obtain that reaction. This may explain why, in the sluggish case just mentioned, hot poultices gave some relief, while passive congestion gave none —in fact rather caused pain.

The lowering of the temperature has already been referred to.

The most important question of all, however, is how are the micro-organisms destroyed and the virulence of their ptomaines neutralised? The germicidal action of the blood serum may explain a great deal, while increased exudation of leucocytes may also be a factor.

In considering the mode of action of passive congestion, it seems worth while to compare with its action the effects of certain other agents or measures upon the same form of inflammation.

The dependent position of an inflamed limb seems always to increase the pain. This may be explained by the action of gravity bringing more blood into the part as well as retarding the exit of both blood and serum from the part.

Ligature of the main artery leading to an inflamed part is often necessary on account of secondary hæmorrhage. During the American War this procedure was found to benefit the inflammation so much that many limbs were saved by the ligature of the main artery as a treatment of the inflammation, even when there was no secondary hæmorrhage. I have been unable to get access to original papers on the subject, but it is referred to in Cooper's Surgical Dictionary (" Inflammation ").

Mr J. Shaw M'Laren has told me of a case of the kind which much impressed him. He was sent for at night to the Infirmary on account of severe secondary hæmorrhage in a bad case of septic inflammation of the forearm. He ligatured the brachial artery, and from that time the arm improved and a good recovery resulted.

In addition to this measure, most surgeons agree in thinking that a free incision into an acutely inflamed part not only relieves the pain after the sting of the incision has passed off, but checks the inflammation and prevents the development of suppuration.

Moreover the application of cold—so strongly advocated by Esmarch—is generally allowed to be beneficial in certain cases and at an early stage—while at later stages of inflammation, when suppuration is impending, hot applications give most relief to the pain and hasten on suppuration.

In all these different measures which affect acute inflamma-

tion—including passive congestion—there may be factors which we are unable to appreciate as yet, but changes in arterial blood pressure seem to be common to most of them. The elastic bandage, a free incision, ligature of the main artery, cold, an elevated position would all diminish the flow in the arteries, while a dependent position would increase it. Certainly warm applications would encourage dilatation of the vessels, but the relief of pain given may be due partly to the serum effused, partly to the drawing off of blood from the affected part by dilatation of vessels near. Warm applications, however, although giving relief to pain and hastening suppuration, often extend the mischief.

As contrasted with these other measuress, passive congestion is immensely superior. It cures the early stages of acute inflammation without allowing suppuration to develop, it checks and limits acute inflammation and suppuration when already advanced, and it soothes pain like an anæsthetic. In fact it acts better than the best of them for any stage of the affection, and it is simpler than the simplest.

References.

Prof. A. Bier. "Hyperämie als Heilmittel," 2nd edition, 1905.

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Meeting VI.—March 7th, 1906

Dr J. O. Affleck, *President, in the Chair*

I. Exhibition of Patients

1. *Dr T. C. Mackenzie* exhibited a case suggestive of GENERAL PARALYSIS. The patient was a youth, aged 19, admitted to the Royal Edinburgh Asylum, certified as being full of delusions. He said that he and his brother had built a house in ten minutes, and that he owned the Empire Theatre; also had delusions of identity. On admission he showed no exaltation of mind; was dull and confused; seemed quite indifferent to his surroundings; gave most irrelevant answers to questions; gave his age as 21, 40, 33; did not know where he was, but said he had been here three days. A few hours later

he exhibited considerable excitement, singing and whistling loudly.

His physical condition was poor; muscular enfeeblement was great; he stood very unsteadily on either foot; Romberg's symptom was not marked; both knee-jerks were much exaggerated; muscles of expression showed fine tremor; speech was much affected, patient hesitating, and slurring his words to a marked degree; tongue was tremulous and showed fibrillar twitching; pupils were dilated and unequal, the right being the larger; reaction to light and accommodation was present. The teeth did not present evidence of congenital syphilis.

History.—Patient was born at full time, and was breast-fed for nine months; suffered from rickets at sixteen months; was five years old before he could walk; was always dull at school; when eight or nine years old began to complain of pain on the left side of his head, this lasting for about one year. Since leaving school he has never been able to earn his living. One year ago he again began to complain of pain on the left side of his head; since then he has had three "fainting turns," as described by his mother, who noticed that after the third he was much confused, and that his speech was very thick. During the last few weeks he has behaved strangely, taking things home which did not belong to him, *e.g.* tins of coffee from a grocer's shop in which he was employed, and selling two programmes for a penny instead of one. He failed to recognise people; became mildly excited, and used to sing a great deal, and early in January 1906 was moved to the Royal Infirmary and thence to the Asylum. Observation and clinical investigation of the patient since admission to the Asylum have confirmed the diagnosis of general paralysis.

2. *Dr Edwin Bramwell* exhibited—

(*a*) A case of SYRINGOMYELIA with a scapulo-humeral arthropathy. The patient, a fireman aged 66, came to Leith Hospital suffering from a whitlow, and was seen by Mr Heron Watson, who had asked Dr Bramwell to examine him. He was an extremely poor witness, and it was therefore impossible to obtain a satisfactory history as to the date of appearance of the first symptoms and their development. For several years he had been unable to straighten his fingers, for some years he had suffered from painless whitlows, and for eighteen months or

more he had noticed an enlargement of his left shoulder and had been aware of grating in that joint on performing certain movements with his left arm.

The diagnostic points elicited on physical examination were: (1) The great enlargement of the left shoulder. The upper part of the humerus and the glenoid region of the scapula were both felt to be markedly enlarged. There was a certain amount of undue freedom of movement at this joint and very pronounced grating. Movement was unassociated with pain. (2) The appearance of the hands, viz., the flexed position of the fingers, more especially of the left hand, with defective extensor power, and the rounded finger tips, and scars indicating previous trophic changes, were very characteristic. (3) The existence of widespread dissociated anæsthesia over the left hand and arm, and over the upper part of the chest on that side.

There was no muscular wasting and neither oculo-pupillary symptoms nor nystagmus were present. There was no spinal curvature, neither was there any indication of involvement of the pyramidal tracts.

(*b*) A girl, aged 20, suffering from a LEFT-SIDED HEMIPLEGIA, which had been present since she was four years of age. The patient had been brought before the Society by Dr John Thomson in 1890, at which time she was suffering from paresis of the left arm, leg, and face, with paralysis of the right third nerve. There was then congestion with œdema of the optic discs, and the child complained of slight headache. There was considerable mental torpor, but there had been no vomiting and no fits. The weakness of the left side of the face had been noticed seven weeks previously, that of the arm and leg three weeks later. The ptosis had been observed a week later still. There was no family history of tubercle. Dr Thomson believed the lesion to be situated in the region of the right crus cerebri, and was of opinion that in all probability it was a tubercular mass.

The third nerve palsy, which was very pronounced at that time, had now completely disappeared, but the hemiplegia persisted.

Dr Bramwell thought that the case would interest the Society, since there seemed strong reason for supposing that there was here a tuberculous lesion of the crus which had long since become quiescent.

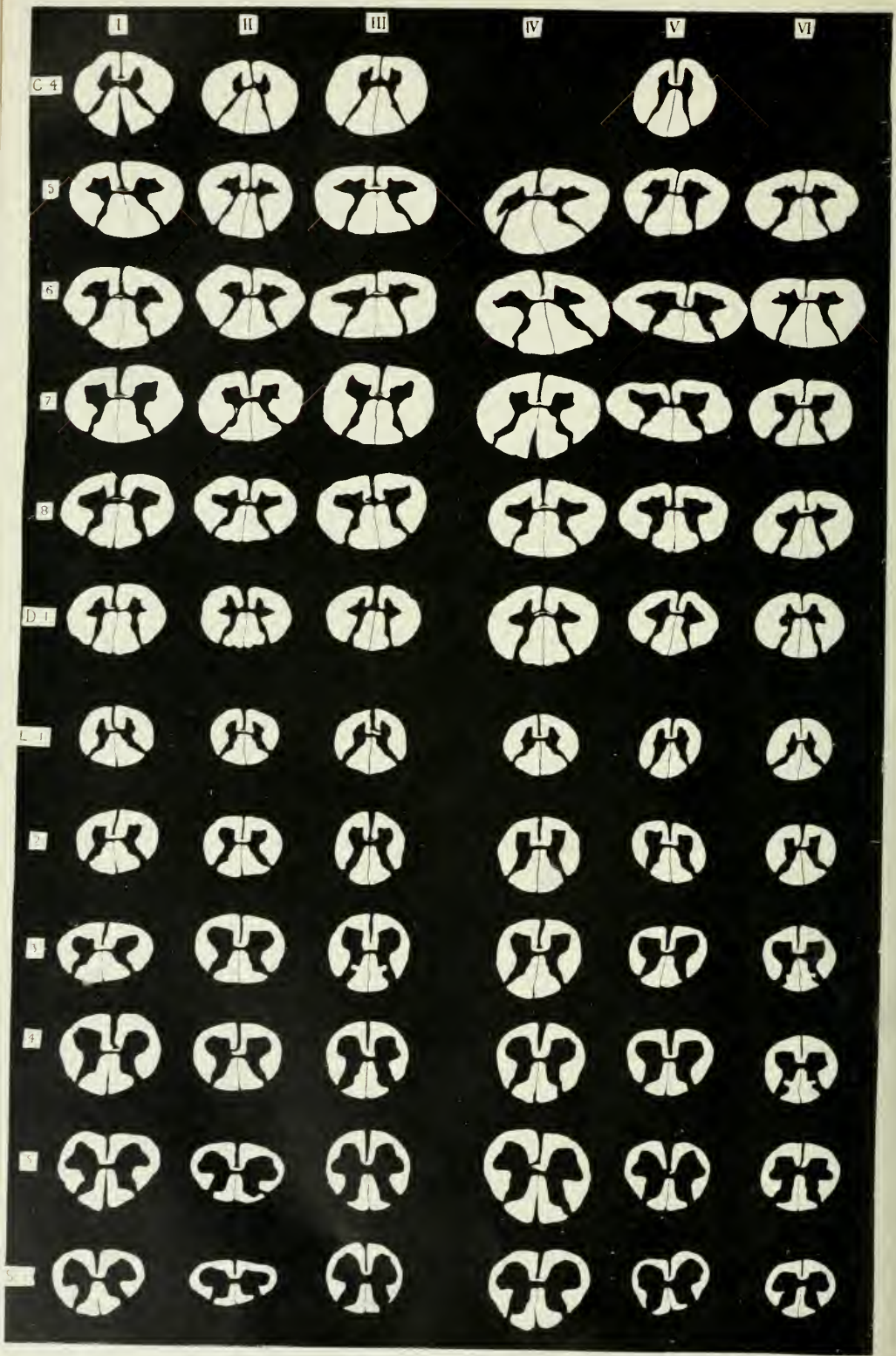

PLATE VII.—OUTLINE DRAWINGS OF SPINAL CORD SECTIONS.

II. Exhibition of Specimens

1. *Dr Edwin Bramwell* exhibited—

(*a*) A series of camera lucida outline drawings from six spinal cords, to illustrate the special features which characterise the shape of the grey matter at the various segmental levels of the cervical and lumbar enlargements.

The method adopted in enumerating the segments was to regard the first small root caudal to the cervical enlargement as the second dorsal. The cords were divided into segments by a series of horizontal incisions at the level of the lowest fibres of each entering root. Each segment was then divided by another transverse incision into two equal parts. The lower portions of successive segments of each cord were preserved in series by a horse hair passed through each portion of cord from above downwards in the region of the left anterior horn. After hardening in Weigert's chrome alum copper solution and embedding in photoxylin, each portion of cord was mounted on a numbered block with its cephalic surface uppermost. A few sections were then taken from each block (*i.e.* from near the centre of each segment) and stained. Outline camera lucida drawings of certain sections (cervical 4 to dorsal 1, and lumbar 1 to sacral 1) were then made. The series was arranged in order and photographed.

The general result of this investigation had been to confirm Dr Bruce's conclusion that there is a type of outline of the grey matter which is characteristic of each segment in the cervical and lumbar enlargements. Space does not permit of reference to the features which distinguish the transverse section at each of the segmental levels represented on the plate (*vide Review of Neurology and Psychiatry*, vol. iv., 1906, p. 344).

(*b*) A series of drawings from two cases of DISSEMINATED SCLEROSIS, illustrating the distribution of the sclerotic patches in the spinal cord. Among points of interest brought out in these drawings were (1) the large area occupied by the sclerotic process in some sections; indeed, at some of the levels represented, the whole transverse extent of the section was involved. (2) A sclerotic area sometimes extended through several spinal segments. (3) It was unusual to meet with a patch of any considerable size which did not reach the surface of the cord. The oval area seen in the posterior columns in several sections

which formed a notable exception to this statement had been, it was interesting to note, figured in more than one of the cases hitherto reported. A number of the larger patches were somewhat wedge-shaped, the base of the wedge being situated at the periphery of the cord. (4) In some of the sections the patches showed a tendency to a symmetrical distribution. This, however, was not nearly such a striking feature in the present cases as in some of those previously recorded. (5) Both the grey and white matter were implicated by the sclerotic process, and although in certain places the patches appeared to stop short at the edge of the grey matter, this appearance did not appear to be sufficiently frequent to justify any dogmatic statement regarding a possible greater resistance to the spread of the sclerotic change through the grey tissue.

(*c*) Photographs showing an abscess of the brain situated in the left occipital lobe, which was located during life from the existence of a right-sided hemichromatopsia. The history of the case was briefly as follows: A man, aged 51, who had been attending for two years the out-patient department of Leith Hospital suffering from bronchiectasis, suddenly developed a very severe pain over the left side of the head. Two days later he had a general convulsive fit with loss of consciousness, and five days after the onset of the headache a similar convulsive attack. On inquiry as to the onset of the fits, the patient stated that before each he experienced a curious sensation, as if a boy was flashing a light from a mirror into his eyes. The light was "a sort of grey colour," and the patient could not recollect from which direction it appeared to come. The flashes, which occurred at first about every half minute, became gradually more frequent, and after they had continued for about an hour and a half the fit came on quite suddenly. He could not recall experiencing any other sensations before losing consciousness. An exactly similar warning preceded the second fit, lasting on this occasion for two hours. These were the only two occasions on which these "flashes of light" were noticed. When examined by Dr Bramwell a week after the first fit, apart from a partial hemianopic defect to the right of the middle line, of which the patient was unaware, nothing objective was elicited. The headache was now less severe, he had had no vomiting, the optic discs showed no neuritis. In particular, there was no word-blindness, no hemianæsthesia, no

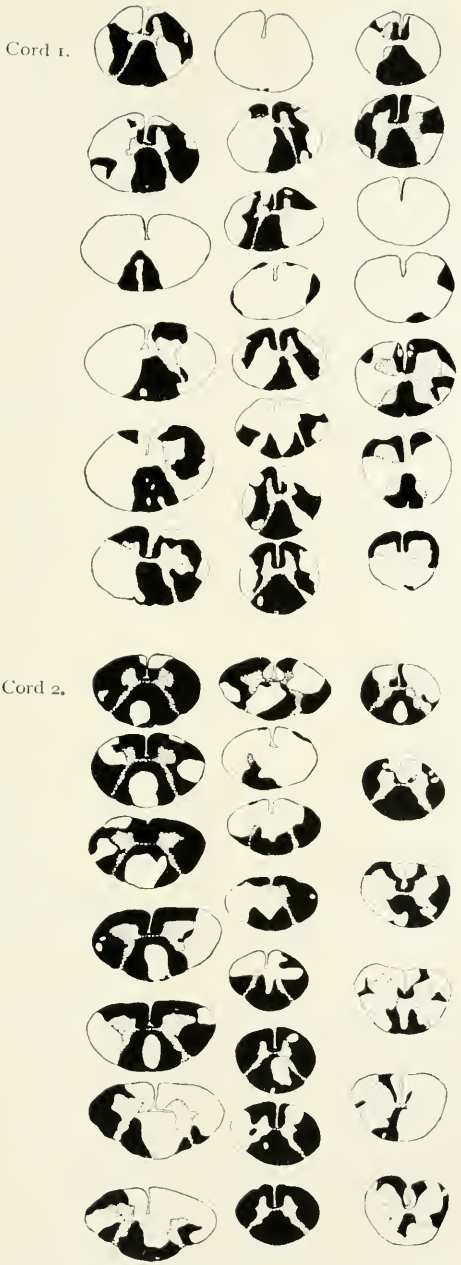

PLATE VIII.—To show the distribution of the sclerotic patches in two cases of Disseminated Sclerosis. (Black=normal white matter: Dotted area=normal grey matter: white=sclerotic patches.)

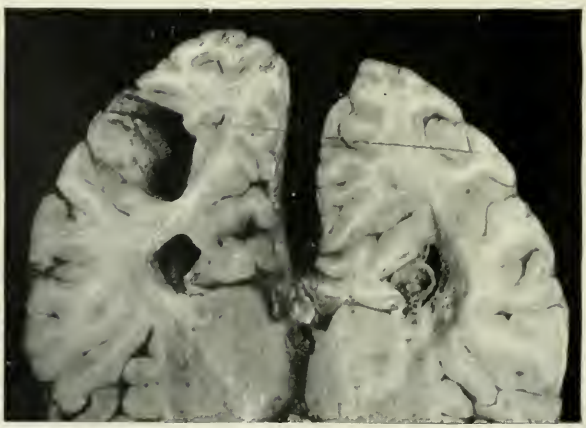

PLATE IX.—Abscess with well-marked capsule in left occipital lobe. In the upper figure the area of cortex destroyed by the abscess is outlined by black dots.

affection of the cranial nerves, no weakness of the limbs, or abnormality of the reflexes. Wernicke's hemianopic pupil reaction was examined for, with a negative result. Dr Sinclair, who made a special examination of the visual fields, found that there was a complete loss of perception of red to the right of the middle line, with loss of perception of white only towards the periphery of the field on that side. On the day after the patient's admission to hospital, indications developed pointing to a gangrene of the lung, and his general condition was such that operation on the cerebrum appeared unjustifiable. Seven weeks after the first appearance of the headache, the patient died, having suffered for three or four days before death from symptoms of an acute cerebral lesion, to which it is unnecessary to refer in the present connection.

At the post-mortem examination, which was performed by Dr W. T. Ritchie, an extensive bilateral bronchiectasis was detected, also an area of gangrene in the lower lobe of the left lung, and a subdiaphragmatic abscess on that side which extended down to and surrounded the kidney. In addition, there was some purulent meningitis.

On cutting the brain after hardening in formalin, the abscess depicted in the plate was discovered. A well-marked thickened capsule surrounded the abscess. The brain tissue immediately around appeared to the naked eye to be perfectly healthy. The abscess had destroyed an area of cortex about the size of a shilling in the anterior part of the third left occipital convolution, immediately posterior to the "Affenspalte." Internally the abscess approached very near to the optic bundle, indeed it would only be possible by a microscopic examination to determine whether these fibres had escaped.

III. Original Communications

1. TRANSIENT BLINDNESS DUE TO SPASM OF THE RETINAL ARTERY

By R. A. Lundie, M.A., B.Sc., M.B., F.R.C.S. (Edin.)

Cases of transient loss of sight, believed from their character and associations to be due to interference with the retinal circulation, are by no means rare, and many references to them are to be found in the literature of the subject (1). The

instances where the fundus has been examined during the attack are, however, few in number.

The duration of such attacks varies very greatly, from a few minutes to hours or even days. It is in the more prolonged attacks, naturally, that opportunities for examination have usually occurred, and it is on their character, therefore, that our ideas of the appearances to be observed are chiefly based. Of these cases the most recent with which I am acquainted, and that most fully described, is one reported by Wagenmann (2), who fully discusses the subject and gives many references to the previous literature.

His patient was a gentleman of 69, who had for two months had frequent attacks of total or partial loss of sight in the right eye, lasting from some minutes to a few hours. In the total attack which was observed, the retinal arteries were found quite empty, and the veins reduced in calibre. After ten minutes, the arteries showed a thin red line of blood ; within half an hour they had resumed their normal appearance, and the sight had returned to its usual condition. Iridectomy was performed ; and for seven months there was complete immunity from the attacks. Then came a severe one, which left the eye almost blind. Wagenmann has collected various similar observations, in one of which the condition was attributed to ischemia of the retina, in one to a passing embolus, in others to spasm of the retinal artery.

Noyes (3) records two cases in which he had observed a similar condition ; in one of these the patient became totally blind in the course of about ten minutes, and remained so for sixteen hours ; twenty-four hours after the commencement of the attack, when seen by Noyes, he had only dim perception of objects (fingers at six feet). The arteries were found very much contracted ; the veins normal. Under the use of amyl nitrite, vision became normal within twenty minutes. Loring (4) mentions that he had " seen several of these cases," in one of which he says that the " attack was at once cut short by the use of nitrite of amyl." He does not, however, distinctly state that he had examined the fundus of the affected eyes during the attacks.

Transient diminution in the calibre of the retinal arteries has also been observed in epilepsy, in migraine, in the cold stage of malarial fever. In some toxic conditions, especially

in poisoning by quinine, great contraction of the retinal arteries is a very prominent feature.

Localised contraction of the retinal vessels has been much more rarely met with. A very curious observation is recorded by Raynaud (5), in one of the cases described by himself of the disease which bears his name. There was partial loss of vision during the intervals between the attacks of lividity of the extremities, which passed off when that condition returned. Ophthalmoscopic examination showed that the branches of the retinal artery were at this time narrower close to the disc than at the periphery; and at times "a sort of partial strangulation" could be seen. There was extremely marked and extensive pulsation in the veins. During the attacks, when the sight was normal, the pulsation in the veins was less marked; but the arteries showed "partial strangulations which, in parts, made them filiform. By exercising a little patience, the observer might be so fortunate as to see these strangulations form before his eyes, last a certain time, then disappear to reproduce themselves on another vessel." These observations, he tells us, were confirmed by Galezowski. I have seen no record of similar appearances in other cases of Raynaud's disease.

The only other case, so far as I know, in which localised transient spasm of the retinal artery has been observed is the very remarkable one described by Benson (6), at the Ophthalmological Congress in Edinburgh, in 1894. The patient was a man of 32, who had for four years experienced recurrent transient attacks of loss of sight. Sometimes the eye affected became totally blind; more often only part of the field was lost. The attacks never lasted more than five minutes. On three occasions the eye was examined during an attack, and each time the portion of the retinal artery corresponding to the blind area was found to be empty for three or four disc-diameters. The bloodless portion of the vessel was seen to creep slowly towards the periphery, and whenever it reached a bifurcation of the artery, seemed to fill up suddenly, so that the fundus again appeared normal.

My patient is a gentleman of 88, with the muscles, including the heart, in a feeble condition. His arteries, though somewhat tortuous, are much less hard than is usual at his age; the heart action is regular and the pulse soft. His

mental power is singularly good; his memory seldom fails him; and his interest in all that goes on in the world is intense. Except for slight chronic bronchitis, no lesion of the internal organs can be detected. The urine contains no albumen or sugar. He suffered last autumn from rather severe lumbago; but this had passed off before the attack I am about to describe. Six weeks after the attack, he had a slight localised dry pleurisy. He was at this time unusually depressed and feeble in consequence of a sudden family bereavement, and was almost entirely confined to bed, where he spent most of his time in reading.

On the evening of the 23rd December last, he was reading, half sitting up in bed, when he noticed that he was not seeing so well as usual. On testing the two eyes separately, he discovered that the left was quite blind.

When I saw him, within half an hour of the onset of the attack, he greeted me with: "It is going off; I can see a little now." I found that the upper part of the field had some perception, but the lower part appeared quite blind.

On ophthalmoscopic examination (it was unfortunately only possible to use the indirect method), I found that the media were clear, the appearances natural, and the vessels normal except the upper main branch of the retinal artery. In this there was an interruption of the column of blood just beyond the margin of the disc, in a section of the vessel somewhat less in length than one disc-diameter. The position of this part of the vessel could be traced as a whitish streak; but it seemed entirely empty. On the proximal side the vessel was of normal calibre; on the distal side the column of blood was continuous, somewhat less than in the other arteries of corresponding rank, but by no means very small. I could almost fancy that a tiny transparent finger and thumb were nipping the artery, just as one nips an india-rubber tube.

I administered a dose of whisky, the only vaso-dilator at hand, and after chatting to the patient for a few minutes, examined again. This time there appeared to be a fine thread of blood in the portion of the artery which was previously empty; but no other difference could be made out. The lower field was still blind.

I left him to obtain amyl nitrite; and on returning was told: "It is all right now; I can see quite well!" On ex-

amination the obstructed artery was found to have recovered its normal appearance. The whole duration of the attack was somewhat less than an hour.

The only explanation possible of the symptoms and appearances described seems to be that there was a localised spasm of the artery. No embolism or thrombosis could have passed away so completely in so short a time ; and there was nothing to be seen in the fundus to suggest the presence of either. The circulation was certainly very weak ; but the appearances were not those found in stasis. No doubt the spasm had affected either the trunk, or all its branches, at the onset of the attack.

Till now there has been no recurrence of the condition, and nothing abnormal can be detected in the fundus, except that the veins are perhaps a little fuller than usual.

I am not able to assign any satisfactory cause for the spasm in this case, and shall, therefore, not spend time in discussing the possibilities.

It is not a little remarkable that among these cases we have evidence of spasm of three distinct types. In most of the cases the whole artery was empty, and refilled gradually. In Benson's, the small empty portion passed onwards by a sort of peristaltic movement, and refilled suddenly. In mine the still smaller empty portion remained stationary, and filled gradually. This variety suggests that there must be much to be learned about the innervation of the arteries, and a wide range of causes leading to disturbance of that innervation.

The spasm in my own case is of a very curious type ; I do not know that such an extremely limited and fixed spasm of an artery has been either observed or suspected. I find, however, in another muscular tube, the intestine, a curiously exact parallel described by Dr Hawkins (7) last January. In this patient laparotomy was performed, and two portions of the intestine were found contracted. A portion of the colon four inches long " was of a pale grey colour, bloodless, strongly contracted and narrowed down to the size of a man's forefinger. The narrowed part passed abruptly into the normal parts above and below. The affected section of the small intestine " (about seven inches long) " presented the same appearance. It was pale, contracted, and narrowed. . . . The line of demarcation of this contracted part from the distended and reddened bowel above and below was sharply defined." (There was satisfactory

evidence that the contraction was not due to any external constricting cause.) We have here a limited, abrupt spasm of a muscular tube, bearing witness to a condition of its nervous mechanism very different from the normal. It is indeed so anomalous that without very direct and convincing evidence we should have been disposed to regard it as impossible. I think the analogy with the spasm of the artery in my own case interesting and suggestive.

From the standpoint of the ophthalmologist, the chief interest and importance of such observations lies in their relation to the cases of sudden loss of sight with contraction of the retinal artery, which were, at one time, all attributed to embolism.

There are many cases recorded as embolism in which the final attack has been preceded by one or more attacks of transient loss of sight, sometimes partial, sometimes complete. In the latter case, the final attack only differs from the others in that the blindness is permanent. Emboli usually have their origin in the heart or the aorta. That successive emboli from such a distant source should all find their way into the same small arterial branch is so improbable that the hypothesis may be at once dismissed. It is, of course, not impossible that there may be a morbid process in one of the minor arteries, *e.g.*, in the ophthalmic, from which successive emboli may be detached and pass into the retinal artery, of which some are so soft and friable that they break down and leave no permanent damage. Such a sequence of events, however, is also very improbable and must be extremely rare. Where prodromal attacks occur, therefore, the final one is very unlikely to be embolic. There are other features described in many of the cases attributed to embolism which make it difficult to accept this explanation of their origin ; but these it would be beside my purpose to discuss.

Even with regard to the pathological evidence, the facts are by no means conclusive. Haab (8), who has made a special study of the subject, goes so far as to say that "in not a single instance, not even in which it was very likely to be an embolus, has the proof of embolic nature been at all convincing."

If we look now at the cases where numerous attacks of transient blindness have occurred, we shall find that in many, as in Wagenmann's described above, in one of Loring's, one of

Nettleship's, and in another I shall recount later, there has been permanent damage at some period following upon these attacks. Loring (9) was one of the first to throw doubt upon the general applicability of the diagnosis of embolism to all such cases; and in one of the cases on which his article is based, there had been attacks of temporary loss of sight in the left eye, sometimes daily, for *twenty-four years*, before the occurrence of one at the age of forty-seven, which produced almost complete blindness of that eye. Nettleship (10), who lays stress upon the importance of heart weakness in relation to these attacks, narrates in one of his cases a somewhat similar sequence.

We may trace a complete gradation from cases where there are numerous attacks of transient blindness without permanent loss, through those where the transient attacks are the prominent feature and permanent damage appears to be only an incident in the history, to those where one or two transient attacks are soon followed by a permanent one, and those finally where there is only one attack, which blinds the eye. Where blindness occurs, the clinical appearances in the final attack are essentially the same, whether transient attacks have preceded it or not. With such a series before us, it is obvious that embolism cannot be the process which has blinded all these eyes; and, even in the last group, very definite evidence must be obtained before this diagnosis can be securely established.

In view of these facts, the definite proof afforded by such cases as my own and others I have mentioned of the occurrence of spasm in the retinal artery is most interesting and significant. When a case is seen at an early stage in which the signs of arterial blocking are present, the possibility of its being due to spasm should be recognised, and vigorous attempts made, by massage, by nitrite of amyl, and other vascular relaxants, to promote dilatation of the arteries. In this way sight may be saved in eyes which would otherwise certainly be lost. In cases of recurrent attacks of blindness of this type, a careful search should be made for any constitutional tendency which may predispose to them, with a view to its correction; and care should be taken to promote very free elimination of waste products (11). Wagenmann (12) urges the performance of iridectomy in these cases, and his own experience, and that of other observers, show that it may be of much value.

Proof of the occurrence of spasm in the retinal artery of

such a degree as to produce marked symptoms has, however, a much wider interest. We know that spasm takes place in the arteries of the retina, because they are open to our inspection. Does it not seem probable that spasm may also be met with in other situations where its presence is not so readily proved?

I have encountered one case which strongly suggests that such spasm really does occur. For part of the notes of the case I am indebted to Dr George Mackay.

The patient is a young lady, aged twenty-two, who has had excellent health and seems to have no organic disease. Her eyes are somewhat myopic (— 3 D), but otherwise appear healthy, with the exception to be recorded. For eight years or so she has had occasional attacks of loss of sight in one eye, which come on without traceable cause, are not associated with headaches, to which she is not subject, last not more than five minutes, and usually pass off completely. There have been perhaps twenty attacks altogether; in all except one the right eye has been the one affected.

In 1901, after one of these attacks, she found that a defect of sight remained, and was examined some days later by Dr George Mackay. The field of the right eye showed a sector-shaped defect in the lower inner quadrant, and the corresponding area of the retina was hazy, swollen, and œdematous. The field remains as it was at that time; the arterial branch supplying the blind portion of the retina is extremely small, and in parts can be traced only with difficulty. Several attacks have occurred subsequently, but none of them have done any further damage.

The appearance observed at the time of the attack which damaged the sight, and the present condition of the fundus, demonstrate that the loss of sight was due to some lesion of the retinal artery. The fact that it was one of a series of transient attacks which left this defect, strongly suggests that the lesion was due to a spasm of the retinal artery, which persisted too long for the integrity of the part of the retina affected. No other hypothesis can link together the frequent transient attacks with the permanent damage to the retina following one of them.

So far the case resembles others that have been reported. But there is one singular feature of the attacks which I have

not yet mentioned, namely, that each of them commences with what the patient describes as "one side of things looking higher than the other," so that there is confusion of objects; for example, uncertainty on which of the two steps she sees in front of her she is to put her foot. This is evidently a vertical diplopia; it is as transient as the blindness, always precedes it, and sometimes seems to have occurred without being followed by loss of sight.

The association of transient diplopia with the attacks of blindness can hardly be explained except by an extension of the spasm beyond the retinal arteries to those of some part of the extra-ocular neuro-muscular apparatus, most probably to some of the arteries in the orbit which supply the extrinsic muscles.

The precise seat of this extra-ocular phenomenon is, however, not material to my argument; what I wish to insist upon is that we have here very strong presumptive evidence that in this case spasm in the retinal artery is associated with spasm of other blood-vessels beyond the eyeball.

The damage to vision which has occurred in this case is itself most instructive. It is another illustration of the fact, already referred to, that these transient amblyopias of vascular origin, however trifling they may appear, are extremely apt, sooner or later, to lead to permanent impairment of sight.

The occurrence of arterial spasm, limited in extent, but sufficiently intense to produce immediate and severe disturbance of the functions of the part supplied, is, I think, proved by such observations as I have recorded; and if it is proved, it has a far-reaching significance.

Cases are not uncommon in which transient, recurrent, and limited losses of brain-function occur, analogous in various ways to the attacks of recurrent blindness which have been discussed. Like the latter, they often prove to be the precursors of permanent damage. Hitherto they have generally been regarded as due to incipient thrombosis; and in regard to their treatment stress has chiefly been laid upon stimulation of the heart. It seems quite probable that some of them, at least, may be due to arterial spasm: in that case, vascular relaxation would be of much more importance than cardiac stimulation (13).

Is it not probable, moreover, that some of the transient "functional" disturbances in other parts of the body may be

due to localised arterial spasm? Pain has been said to be "the cry of a starved nerve for food": it seems not unlikely that some other paroxysmal pains besides angina pectoris may be found to yield to the influence of vascular relaxants.

I have ventured into a region which has been but little explored, but on which more light may be expected from future research. May we not hope that farther observation of the anomalies of the retinal circulation will help us to a better understanding of the processes of blood-supply throughout the body?

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2. VACCINE TREATMENT IN SURGERY

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It is fitting that evidence on the effects of new methods of treatment should be heard from many sides before such are extolled or condemned. Still better is it that such methods should be tried personally if the principles on which they depend are shown by sensible and reliable workers to satisfy a scientific conscience. About two years ago I heard such evidence with regard to vaccine treatment, founded on Professor A. E. Wright's excellent investigations, and, being satisfied with the authority, have during the past eighteen months endeavoured to test such treatment clinically in as fair a way as I could. Hence I have ventured to appear before you to-night to record my experience. I wish to lay facts

before you, and therefore, where possible, I have had my statements verified by others who have seen the cases. Before putting these facts before you it may be well once again to run over the principles underlying the method of treatment.

By vaccine treatment is meant, roughly, the injection into the patient of quantities of the organisms (dead) which produce the disease. It is found that the resisting power of an individual against the specific organism can be raised with safety by such means. In chronic localised infections or even in some general infections of a chronic character—*e.g.*, Malta fever—sufficient stimulus to successful resistance is wanting. To use such a vaccine during an acute disease would be simply adding fuel to the fire. After such vaccine is injected there is produced a depression in the resisting power—a negative phase—which lasts for a variable time according to the amount or virulence of the vaccine. Following on the negative phase the resistance rises till it is in most cases above what it was before—a positive phase. By repetition of such doses at suitable intervals a cumulative positive phase is usually obtained, so that resistance is much increased. If, however, a second injection is made during the negative phase, this phase is augmented so that harm is done. Hence the necessity to estimate the opsonic power of the patient's serum, which forms a convenient indication of the resisting power, to know when the positive phase is well developed, as then is the time to give another dose. I wish to indicate at this point that if the cycle of events following the injection of a specific vaccine has been estimated a sufficient number of times in different individuals, so that one knows the average time required for the positive phase to develop fully, in my opinion it is unnecessary to estimate the opsonic index in most cases. On this idea I have worked for the past seven months, and I think it a matter of importance that my results have not suffered therefrom.

Tuberculous disease in its various phases has provided the most frequent test for the efficacy of vaccine treatment. Koch's tuberculin R. differs from the other vaccines used in that the most soluble constituents of the culture are abstracted from it. I have always employed that supplied by Meister Lucius and Brüning, Höchst, Germany. For brevity I shall

call it hereafter T.R. It will best serve the purpose to describe the effects of T.R. treatment on the different lesions.

Tuberculous glands.—If these are simply enlarged—not broken down, caseating, or calcified—in every case I have found that the glands disappear after a very few injections of T.R.; in quite early cases no trace of enlargement remains after even only two or three injections. This has been my experience in three cases. One must bear in mind that a surgeon does not usually get such early cases, hence my experience is limited. It is probably impossible to say at first whether caseation has occurred in any given gland, but if the gland, of consistence usual in such cases, has not decreased in size markedly after from six to eight weeks' treatment, I have always suspected caseation, and at operation this condition has been found. In many of the cases one could compare two or more glands of apparently equal size and consistence. During treatment one or more would so decrease in size as to be impalpable, while the caseating ones would decrease only to a slight extent. If periadenitis, with consequent matting and tenderness, had occurred, this usually speedily cleared up. The tenderness disappeared and the glands became more defined and movable, then smaller in size. If caseation has occurred, operate. Operation need not be vigorous. I have such faith in T.R. that I am going back from my previous attitude in the treatment of tuberculous glands. I used to think that thorough excision, where possible, of all tuberculous foci was the only rational treatment. Now I find that in, *e.g.*, *isolated* caseating glands, gentle scooping out of the caseating core is sufficient. If a depressed cicatrix results, this may be excised by a very small incision under a local anæsthetic at a later date when the patient is quite sound. If a *mass* of caseating glands exists, then excise *en masse* in the usual way. Somewhat similar remarks apply to broken-down fluctuating glands. At first I tested the capabilities of T.R. to cause absorption of the fluid material, but in no case did it succeed, although in all the "abscesses" became more circumscribed; if tenderness were present it disappeared, and if reddening of the skin were present over the gland, this lessened or disappeared also. Had fluid been absorbed, the condition left would have been at best similar to that in the caseating gland. Hence I think it is waste of time to delay operation long in

such cases. In one case I aspirated the fluid contents of the gland capsule, and gave an injection of T.R. Where the aspirating needle had been inserted, a sinus formed, which healed after a second injection, and after a third there was no trace of enlargement. I think that post-operative healing is without doubt promoted by the giving of one or two injections of T.R. before operation, which should take place about the time when a third injection is due. The patient's resistance is higher and the general health is better, hence healing is quicker and operation need be less extensive. In *isolated* suppurating glands a small incision, varying in size according to the depth of the gland, with gentle curetting, is all that is required. After two days' packing with gauze the incision may be sutured, if necessary, without bad effect. If caseating glands are studded around the softened one, it is better to excise the whole affected mass. If the glands are calcified, excision is to my mind the only course. One cannot expect T.R. to cause absorption of cretaceous material, and one ought to get rid of a source of danger to the patient. If the glands have broken down, and infiltration of the skin, rupture of the abscess, formation of sinus, and infection of the focus by other organisms taken place, I have found that simple T.R. treatment does not suffice in many cases. Either thorough "scraping" or excision of the affected parts should be carried out, or else the suitable vaccine or vaccines besides T.R. should be given. One or two injections of streptococcus, or staphylococcus, or of both, should be given. With T.R. alone such cases often improve only to a limited extent, and then show no further improvement, in spite of variation in the dose or time interval of injection. Of course, the usual precautions which accompany ordinary surgical treatment should be carried out—*e.g.*, removal of bad teeth, enlarged tonsils or adenoids, or other source of infection. Fixation of the part will promote healing of this, as of other tuberculous affections. To my mind, foul discharging sinuses and hideous scarred necks resulting from advanced tuberculous disease, should become unknown. The disease should never be allowed to reach that stage.

Tuberculous joint affections.—Here, again, the prospect is a hopeful one. But ultimate complete success in restoring full healthy function will be obtained only by making a correct diagnosis in early stages and adding T.R. injections to those

well proved and approved lines of treatment usually carried out. All the same, one of my joint cases, tuberculous arthritis of the knee, has been completely cured by the employment of T.R. alone. Since the injections were begun the patient has had no splinting or any special local treatment and no other nternal remedy has been given. At the commencement of the T.R. treatment he had a sinus, very recently formed, over the internal condyle, which was much enlarged. There were thickening of the synovial membrane, effusion into the joint cavity, pain, and much limitation of movement. The joint is now practically normal to appearance, and movement has increased by about 45°. The patient has no pain and can run about freely with only the slightest limp. All the same, I recommend most strongly that all treatment which will favourably influence the course of the disease should be employed. One cannot afford conscientiously to run risks with such patients. To my mind, however, a new element has arisen in the treatment. Previously, when a patient was (to even a slight extent) the subject of night-starting, shrieked with pain at any attempt made to move the joint, and when effusion into the joint and thickening of the synovial membrane were present, one gave, to say the least of it, a very guarded prognosis as to the future of that joint. It *might* disintegrate completely, and the most favourable outcome was that the disease would subside, but a stiff joint, in more or less good position, was practically certain. Operation cures the disease to be sure in most cases, but in too many it leaves much to be desired with regard to function. In the lower extremity it is practically essential to procure firm ankylosis. I feel certain that the outlook will be far more cheerful when the early use of T.R. is made. I have mentioned one case of arthritis of the knee. I have under treatment a case of tuberculous synovitis of the knee following displacement of the inner semilunar cartilage. Microscopic examination confirmed the diagnosis of tuberculous disease. The knee measured two inches more in circumference than the sound one. Movement was limited to about 30°. In such a case, in the event of healing under ordinary treatment one would have expected dense adhesions, but the patient is obtaining freer movement as the joint is rapidly regaining its normal size. The condition was becoming steadily worse before T.R. was given. A third case of hip-joint disease showed the

symptoms which I have already indicated (night-starting, pain on handling, and especially on attempted passive movement, etc.). Here I have the advantage of having had my observations confirmed by one of yourselves, who gave a guarded prognosis, except that he was quite certain that even the most favourable outcome would be a stiff joint with shortening. With this opinion I was practically agreed. I have no doubt that he will be pleased to hear that the little patient can wriggle about in bed like an eel, can sit almost bolt upright without any sign of discomfort whatever, and allows one to handle the joint without demur. I venture, therefore, to think that such joints should not be absolutely immobilised for the long period which has hitherto been customary. By so fostering adhesions we may prevent what would otherwise be a perfect functional result. If not contra-indicated by the general progress of the case and the tendencies of the patient I suggest that about from two to four weeks after all tenderness on palpation of the joint has disappeared gentle passive movements be attempted every few days, and that the patient be allowed well-governed freedom to move the joint if inclined. If any sign of deterioration occurs renew the stringent precautions for another month. Thus I believe that the course of treatment will be shortened by about half of what has hitherto obtained. It is interesting that in a case of resection of the shoulder for tuberculous disease 18 months previously, where the cervical glands in the posterior triangle had recently become enlarged to the size of a hazel nut, the patient stated spontaneously, after a couple of injections of T.R., that movement at the shoulder had become much freer. The glands (one suppurating, aspirated) became impalpable after the third injection. She had a very bad tuberculous family history.

There is often difficulty in determining whether a deep joint, such as the hip, is really tuberculous. One can come to a decision by giving a big dose of T.R. in order to provoke the local reaction which will occur if the focus be tuberculous. This is very undesirable, because one may thus lower the resistance (or, in the language of the opsonist, produce such a decided negative phase) so that the process, instead of being a very chronic one, may light up and prove very difficult to master. It is much better, although not quite so trustworthy, where it is possible to examine the blood only, to ascertain the

opsonic index to tubercle bacilli. According to Wright and many others this is of much clinical value. If it be low or unduly high one should at least be very suspicious of tubercle.

A young woman consulted me about her wrist, which was giving her much pain and preventing especially her piano playing. There was slight thickening of the proximal end of the fourth right metacarpal bone. This disappeared after fixation of the wrist in plaster-of-Paris for some weeks, but the pain persisted and became worse as time went on, and there was a suspicion of recurrence of the thickening of the bone. X-rays gave no help. The opsonic index to tubercle bacilli was 0.75, so I began T.R. injections, with the result that the trouble rapidly disappeared.

A very different picture from the point of view of rapid results is presented usually by cases where abscess formation with subsequent development of sinuses and compound infection has occurred. In none have I obtained satisfactory results with T.R. alone unless after operation, and in the majority, although great improvement has occurred, yet it has been slow and subject to occasional retrogression. Here, however, I have not many cases to judge from. One case already recorded was a boy from the poorhouse, with elbow, wrist, metacarpal, and ankle much disintegrated—to say nothing of far advanced disease in his cervical glands. The rapidity with which his lesions healed was very striking. The case of a little girl, in very poor general condition, who came to hospital with a profusely discharging sinus in front of her left hip-joint, is probably the next best in result. The head of the femur was far gone, and there was a rather large cavity, filled with pus and lined with very unhealthy granulations, in front of the joint. The bone and cavity were thoroughly scraped. She healed slowly with T.R., but made rapid strides when staphylococcus vaccine was given as well. There remains now only a very small superficial ulcer. In a man, aged 45 years, a very advanced case of hip-joint disease, the result has been unsatisfactory. He was, however, in bad general condition, suffered from incipient waxy disease, which caused troublesome diarrhœa, and he persisted in taking unsuitable food. To give him a chance of life I undertook treatment. The head and neck of the femur were rotten, and there was besides necrosis of the innominate bone. Numerous foul cavities

radiated in various directions from the diseased bone. Several operations were performed. He would not listen to proposals of amputation. After T.R. was given he improved only to a limited extent, and then lost ground in spite of variation in dose and time intervals. His opsonic index averaged from 0.75 to 0.8. He improved much more when mixed vaccines of staphylococcus and streptococcus (which organisms were found in the discharge) were used. In fact, only then did any satisfactory healing occur. A large granulating area which had formed in the line of an extensive posterior incision healed with the exception of four sinuses. This area had remained unaltered for weeks. The patient has tired of hospital and gone home, and one cannot say that he is better.

I think you will agree after hearing details of these cases at varying stages that it is now even more desirable to prevent, as I believe one can prevent, the very miserable local and general condition of advanced and progressive cases and the disablement and too frequent deformity of cases where cure has been effected. This can be done only by accurate diagnosis and by prompt and rigorous treatment in the early stages. I have no doubt that the very much brighter outlook in these cases will soon be appreciated by those who add T.R. injections to their other treatment.

Tuberculous peritonitis.—In this class I have only one case to record. A hospital nurse, with a very bad family history, developed what turned out to be severe tuberculous disease of the ovaries and tubes, with accompanying peritonitis in the lower part of the abdomen. She had previously a sharp attack of pleurisy with effusion. There were complications, non-tuberculous, which hastened operation. Her right tube and ovary were removed, and owing to the conditions it was necessary to leave a tampon in the pelvis, the ends being brought out at the lower part of the abdominal wound. The patient was very ill for many months after the operation, and was making a very tardy convalescence, if convalescence it could be called, until injections of T.R. were begun. There was then a most decided change in a week or two. She gained appetite, flesh, and strength. The abdominal wound, which had become a large, scooped-out, unhealthy, granulating area, healed until only a small sinus was left. This led into a cavity in the pelvis, which was then drained per vaginam.

There is still a very tiny suprapubic sinus. She is now in comparatively robust health, and capable of taking a fair amount of walking exercise. Six months ago every one in connection with the case thought that the prognosis was well-nigh hopeless.

Psoas abscess.—Either aspiration or incision is necessary at some part of the treatment of these cases. Personally, I prefer incision, so as to be able thoroughly to evacuate caseous material and pieces of diseased bone, either movable or fixed. I believe that energetic scraping of the abscess wall will be obviated by the use of T.R. I have, however, not had an uncomplicated case of tuberculous psoas abscess to deal with since I have become convinced of the efficacy of T.R. I have to report two cases where multiple infection occurred. One was an anæmic, sallow-looking youth, who informed us at the hospital that during the previous week his trousers had rubbed through the skin over a very large swelling in the left groin and upper part of the thigh. A great quantity of pus had escaped. There was a hole in the skin of about one inch in diameter through which masses of caseous material and thin flaky pus were still escaping. The unhealthy edges of the hole were excised by an elliptical incision, and the cavity below Poupart's ligament was scraped out. Through an incision above the iliac crest the upper part of the abscess was opened. Several pieces of loose bone were removed, and the cavity was scraped in a perfunctory way compared with my usual practice. Both wounds were sutured. All the stitches gave way, and for a couple of weeks free discharge occurred. Ten days after his operation T.R. injections were begun. Later he got mixed staphylococcus and streptococcus besides. The granulating wounds kept very healthy and healed rapidly, discharge from the depth soon ceasing. His general appearance and health are vastly improved. The other, a weakly emaciated girl aged 17 years, came to hospital with a sinus in the groin, from which fairly profuse discharge had escaped for several weeks. Otherwise the case was similar to the one already given. T.R. injections had a beneficial effect, but far greater improvement resulted when streptococcus and staphylococcus vaccines were used in addition. The wounds have practically healed. Infected psoas abscess is a condition of evil repute, but these cases show that one need not be so hopeless nowadays about

the prognosis. Judging from my own limited experience I should have given a bad prognosis in both cases with ordinary treatment.

Tuberculous disease of upper air passages.—A case of rhinitis, with typical tuberculous ulceration of mucous membrane, and caries of turbinates and bones of the bridge of the nose, with secondary suppurative infection of the absorbent glands, had been treated over several years by all sorts of local manœuvres with only temporary benefit. The patient came to me again about the middle of January, saying that she thought that another operation must be necessary, and was afraid that the disease was going to break through the skin at the bridge of the nose. One injection of T.R., supposed to be $\frac{1}{6\,0\,0}$th of a milligramme, produced considerable reaction, lasting for a week. A second smaller dose, with staphylococcus added, was given after 18 days, and repeated at intervals of 14 days. The patient is now almost cured.

Tuberculous teno synovitis.—A young patient, with a bad family history, was operated on by me for tuberculous synovitis of the common flexor sheath of her right hand. This was followed by perfect healing, but 18 months later the flexor sheath at the base of the middle finger became similarly affected. One injection produced markedly beneficial effect, in spite of the fact that the movements of the finger were controlled only voluntarily—*i.e.*, no splinting was applied. She was cured after three injections.

Genito-urinary tuberculosis.—As I have taken up too much time already, I do not wish to say much about this, except that I have not obtained the constant and progressive good effect from T.R. alone recorded by other observers. In fact, had I begun by trying T.R. on this class, I should not have been here to-night, as I should have had so little to record. I am convinced that it is advantageous at an early stage to obtain information as to the nature of the complicating organisms, for in such cases complication takes place early. Then make and use the suitable vaccine. In uncomplicated cases, or where purely tuberculous deposits are left behind—*e.g.*, in the prostate or the vesical end of the vas deferens after removal of the testicle—T.R. causes more rapid absorption of such deposits than usual.

The use of other vaccines.—Of the effects of other vaccines

on their specific diseases I do not intend to say much. I have not so much experience of their use, and the results have not been so uniformly striking in cases where the infection was uncomplicated.

The usefulness of other vaccines in conjunction with T.R. in mixed infection of tuberculous foci has been already and probably sufficiently indicated. The same degree of usefulness may evolve in other conditions. It is well known, *e.g.*, that once a gumma has "broken" through the overlying skin or mucous membrane, it is frequently infinitely more difficult to bring about cure with ordinary medication than it would have been before ulceration occurred. It is a case of mixed infection again. An example of the good effect of staphylococcus and streptococcus vaccines occurred lately. The patient's back was the seat of several ulcerated syphilitic lesions. Two large areas were covered with rupioid crusts, and over the erector spinæ opposite the twelfth rib on the right side, was a scooped-out ulcer, dirty, sloughy, discharging freely, and smelling badly. It measured two and a half inches across and one inch in depth. All sorts of treatment had been given to these parts by numerous skin specialists in different parts of the country, but during the past year they had become rather worse than better. Injections of staphylococcus and streptococcus produced an immediate improvement. The affected areas became cleaner, the granulations more vascular and firmer, and the surrounding parts much healthier and more natural looking. I do not think that healing will occur without operation. Whereas I had scruples about operating from fear of making matters worse, I now have none.

Staphylococcus and streptococcus vaccines are of use in another class of diseases, ulcerated cancers. Prompted by Mr G. L. Cheatle's suggestion I have given these vaccines in three cases of extensive operation in the mouth, with the idea of preventing septic pneumonia. In one patient, aged 65 years, the extent of the epithelioma required removal of the whole of the horizontal ramus of the lower jaw on one side and of the adjacent part of the floor of the mouth and cheek, besides the glands of the anterior triangle. The patient was a weakly, anæmic individual, of a type prone to succumb to pneumonia. In the other cases, one, a case of sarcoma in a patient, aged 45 years, and another, a case of epithelioma in a patient aged

69 years, involved, curiously enough, in both patients the removal of the back part of the upper part of the jaw (behind the first molar tooth), the tonsil and adjacent parts of the pharyngeal wall, palate, and tongue, and all the tissues between these and the ascending ramus of the upper jaw. In these cases I consider that septic pneumonia was peculiarly liable to occur. In none of the cases was there ever the faintest suspicion of lung trouble. It would be a great matter if such treatment obviated the necessity of tracheotomy. The injections were given from five to seven days before the operation. The first patient had previously two other injections at weekly intervals. What impressed me was the shrinking and cleaning of the affected areas, especially in the first case. This patient expressed the opinion that operation would be unnecessary if the improvement continued. His mouth was foul from the presence of many carious teeth. The sockets left after extraction previously to operation healed more cleanly and quickly than usual. In this cleansing effect, then, we have another reason for the use of these vaccines before operation in such cases. There are less sloughing and less discharge during the after-treatment, and therefore less trouble in keeping the parts sweet, and, most important, less danger to the patient. Apparently this beneficial effect begins to diminish after from ten to fourteen days. Injections repeated after the operation will prevent this probably, as evidenced by one of the cases. I intend to widen the sphere of usefulness, and make similar injections before such operations as for cleft palate, so as to increase the probability of primary union. Other possibilities crowd into one's mind—*e.g.*, for the prevention of peritonitis as the result of operation for closure of fæcal fistula (here bacillus coli might be given as well), or previously to operations on diabetics or "albuminurics," who are so prone to the onslaughts of septic organisms. In cases of severe accident wounds, although by early thorough mechanical cleansing and clipping away of lacerated structures one procures usually perfect healing, it may be possible to improve statistics further by giving before the operation vaccines of the organisms of suppuration. Where such thorough cleansing has been done I find that when suppuration does occur it occurs at about the end of from seven to ten days, and is usually mild in character,

so that the positive phase following the injection of the necessary vaccines will have developed several days earlier than this, and may prevent such suppuration. The preliminary negative phase might, however, foster the earlier development of the infecting organisms, so before passing any opinion one requires to compare many cases observed by trustworthy men.

In the pure streptococcus or staphylococcus infections one does not require to, one had better not, use vaccines in the early stages. But for the bursting of an abscess, or the use of the knife, all such cases should, theoretically, according to this resistance theory of Wright, prove fatal, for is there not a continual supply of poison absorbed into the system, and should there not, therefore, be a continuous increase in the negative phase? Why should erysipelas become arrested? Why should there be a crisis in pneumonia according to these ideas? I shall not attempt to answer, but merely say that theories do not fit all phenomena where living tissues are concerned. Even though such abscesses are not relieved, they sometimes dry up, and this is more likely to happen the nearer they are to the surface of the body. Certainly the small circumscribed furuncular abscess frequently does. Evidently there is a victory for the tissues and fluids concerned. But yet there is not sufficient resistance provoked, for how often do we see crop after crop of such boils coming out, frequently over a period of months. Here there is a clear indication for the raising of resistance by injection into the general circulation of the specific vaccine, and it has been done repeatedly with success. In *chronic* septicæmia or pyæmia, again, cure will probably be obtained by giving a fillip to the resisting powers by the injection of the suitable vaccine.

One can only indicate probable usefulness for gonococcus vaccine in *chronic* gonorrhœal infections, such as gleet, prostatitis, epididymitis, endometritis, salpingitis, arthritis, and so on.

In a case of pyelitis, where internal remedies, prolonged drainage, with daily flushing of the renal pelvis, and plastic operation on the deformed pelvis, had failed, I tried vaccine treatment. The vaccine was made by Professor A. E. Wright, from the culture obtained from the patient's urine. It was a

pure one of bacillus coli. The case was evidently proceeding rapidly to cure, when an attack of influenza occurred, and no efforts would induce the number of bacteria in his urine to decrease below 1,000,000 to 2,000,000 per cubic centimetre. All the same, the average number was at one time about 30,000,000. The progress of this case has been checked by the patient's opsonic index to bacilli coli.

Before proceeding further I should like to say that I would fain avoid technical details in connection with our subject, my own inclinations being strengthened by a hint from your secretary, but one cannot altogether disregard them. I do not wish to refer to the opsonic index problem further than to discuss the question whether such an estimation is necessary for carrying the great majority of cases to a successful issue. I assert that it is not. If my assertion is wrong, it means that many cases deserving of the treatment will have to do without it on account of the expense and labour involved in the calculations. An ordinary practitioner cannot possibly afford the time necessary. Even if he could, unless he has passed through very special training his observations will be of no value. The average patient cannot afford to pay for the services of the skilled bacteriologist as well as those of his own medical man. One must bear in mind that a great bulk of patients of the non-pauperisable working and middle classes are outside the benefits of what may obtain in hospitals. As the usefulness of the treatment is appreciated, and its application extends, even hospital authorities may grudge to pay for the amount of skilled labour involved if clinical symptoms are not sufficient to guide one during the course of treatment. I assert that accurate clinical observation is quite sufficient, but would repeat that thorough experimental investigation of the effects of any specific vaccine must precede its use clinically in this way in order more easily to arrive at the proper approximate doses and time intervals of giving these. The opsonic index is necessary for the investigator, but not for the clinician in the majority of cases. My cases prove the point very satisfactorily, and I think that nothing more need be said by me. One cannot do without the services of the bacteriologist, however. He must find out for us what the organism is which is causing the disease, and prepare the corresponding vaccine.

It will be best to consider now what are the proper dosage and time intervals at which the doses should be given. As stated, these must first be ascertained approximately by the bacteriologist. I do not think that one can be dogmatic about dosage. From my experience in this line I should say that one of the important things is *to get rid of the inclination to increase doses* so as to produce better results. Not very long ago Professor Wright was kind enough to send me a list of the doses of a few of the vaccines which he had been giving. They were as follows: Staphylococcus, from 500,000,000 to 1,000,000,000; streptococcus, from 100,000,000 to 250,000,000; gonococcus, from 50,000,000 to 250,000,000; bacillus coli, from 100,000,000 to 1,000,000,000; and tuberculin R., from $\frac{1}{2400}$th to $\frac{1}{600}$th of a milligramme. I should say that one should give even less than these. For example, I have been getting very marked results from the use of about one-sixth of this amount of staphylococcus vaccine at a time. As Professor Wright says, it seems well also to prolong the interval and to lessen the dose of vaccine in the later stages of treatment.

The dose of T.R. which I have been giving usually is $\frac{1}{300}$th of a milligramme for either adults or children beyond the age of from three to five years. Children seem to be able to stand relatively more than adults, although I cannot say that the good results have been more striking or rapid. The doses have been repeated at intervals of usually ten days, lengthened to about fourteen days if so indicated by the clinical symptoms. After two or three months repeat only every three or four weeks. After the performance of any extensive operation, or one in which vigorous scraping has been done, it is better to omit the dose until from ten to fourteen days have elapsed, because a negative phase is produced by such operations. For the same reason I do not give a dose for a few days after the patient has arrived in hospital, especially if there is much discomfort about the affected part or rise of temperature. It would appear to most to be quite a simple matter to dilute the solution of tuberculin to easily manipulable strength, but this is not an easy thing to do accurately and aseptically. I prefer to get it done by the trained bacteriologist. I have reason to say that accuracy in this matter is important. As I have indicated, doses of from 50,000,000 to 150,000,000 of staphylococci

seem to me to be quite sufficient. I have given them at intervals of from five to eight days before operation, and at similar intervals in ordinary curative treatment. From 50,000,000 to 100,000,000 of streptococci have been given at similar intervals in similar circumstances. Bacillus coli gives, I should say, best results in doses of from about 50,000,000 to 150,000,000. In my experience doses above that are likely to produce severe general reaction. In giving mixed vaccines it is apparently immaterial, so far as T.R., staphylococcus, and streptococcus are concerned, whether they are given together or alone. Apparently each works quite independently of the other. This fact has also been proved experimentally. The nearer to the surface of the body these vaccines are injected the more likely are they to produce an intense reaction at the point of injection. This refers especially to staphylococcus and streptococcus vaccines. Therefore it is best to inject into the belly of a fleshy muscle and to make the patient move the part so as to dissipate the dose. It is well that the patient should not indulge in exercise for some hours after the injection.

Now comes the question, what are the clinical symptoms by which one can be guided reliably? One should give a dose which will just fall short of producing a definite local or general reaction. If larger doses are given too great a drop in resistance may be caused and harm result by providing freer scope for development of organisms previously more or less inert and under control of the patient's defensive system. Witness the results in the days when tuberculin was first used! If too large a dose be given the patient may have discomfort or actual pain, swelling, and other increased inflammatory signs in the part affected. General signs which I have noted are headache, loss of appetite, malaise, nausea, even vomiting, rise of temperature, aching bones or joints, and in the cases of streptococcus and bacillus coli severe rigors have occurred.

If the vaccine be doing good the great majority of my patients have experienced improvement in the part affected and have shown and expressed marked improvement in general health, their appetite and sleep have improved, they became brighter, and they put on flesh. For a day or two following the earlier injections there may be such slight signs as increase

in discharge, or discomfort to the extent of more active consciousness of the part affected, but as the positive phase develops the discharge lessens, the swelling decreases, the pain and tenderness tend to disappear, and so on.

It may be unwise to appear so hopeful about the future of cases which are amenable to vaccine treatment. With reference to tuberculous cases, at first, of course, I had to test the treatment for myself, and this had to be done in the less important cases. I began the treatment in a somewhat sceptical frame of mind. When I was convinced of its utility I tried it in all my tuberculous cases, and then had to go a step further and add mixed vaccines to T.R. in infected cases. All this took time, hence the number of cases recorded which are still under treatment but which I am convinced are going on to complete cure. The general and local conditions in these are very much healthier than in similar cases after the same length of treatment without T.R. or other vaccines. I have recorded the effects of the treatment on patients in different stages of disease as faithfully as I could without undue length. In a large number of the cases (out-patients and private patients) the surrounding conditions were the same before and after T.R. treatment was begun, and no other internal remedies were given. My experience leads me to say that the earlier the stage of tuberculous disease the more certain the cure. Perhaps even a majority of such cases will heal by other means, but I think there will be a greater certainty of cure and less likelihood of impaired function if T.R. be used in the proper way. I draw attention particularly to the value of T.R. in preparation for operation.

If I may be permitted to trespass on medical ground I should like to say that I think pulmonary tuberculosis is comparable, so far as treatment is concerned, with tuberculous disease elsewhere. Of course, we know how favourably early cases of phthisis are influenced by modern methods of treatment. By the addition of T.R. to these I think that cure will be much more certain. If the case is so advanced that purulent expectoration is present, then organisms other than the tubercle bacillus are at work, and they require the use of mixed vaccines just as surgical cases do. I believe that, as in surgery, only occasionally will such cases be cured by T.R. alone. Cases

with high temperature, profuse expectoration, great wasting, excavation, and so on are similar to the advanced case of hip-joint disease which I mentioned, and whose life has, to my mind, been merely prolonged. These phthisical cases, unlike the majority of surgical ones, cannot be investigated by sight and touch, nor can we estimate with the same degree of certainty the amount of local reaction following injection. Hence it would appear more desirable to be guided in such cases by the opsonic index.

I think it reasonable that T.R. should be given as a preventive of recurrence. I mean in future to give, say, three injections during one month out of every six until the patient is considered safe.

You will have appreciated the fact that want of progressive improvement during T.R. treatment is, in my experience, almost an indication for operation, apart from other considerations. In the case of tuberculous glands it is so, and it would appear to be the same in joint disease, for where purulent flaky effusion, caseation, or necrosis of bone is present T.R. causes only partial improvement.

As Wright and Bulloch have pointed out, the good effect of Bier's passive congestion on localised infections may be explained in that it forces into the focus fluid richer in resisting substances than those present in the infected area. The same thing happens when an abscess is opened.

The whole subject of vaccine treatment is of very peculiar and not unequivocal interest to the active surgeon, because its success means the removal of much work out of his hands into the hands of the general practitioner, or rather that the means of treatment at the disposal of the general practitioner will be such that there will be no necessity to hand so many cases over to the care of the surgeon.

I must now apologise for having taken up so much of your time. There are many other thoughts which have occurred to one during the study of the cases I have put before you, but these require to be tested before being presented in public.

3. OCCUPATION MORTALITIES

By James C. Dunlop, M.D., F.R.C.P. Ed., Superintendent of the Statistical Department in the Office of the Registrar-General for Scotland

The detail of the study to which I desire to draw the attention of the Society is published in the Decennial Supplement to the Forty-eighth Detailed Annual Report of the Registrar-General for Scotland. It is based on the number of male persons found by the 1901 census to be engaged in the various occupations, and on the number of deaths registered in Scotland during the years 1900, 1901, and 1902. The reason why the deaths of three years were used for this purpose was to make the field of observation as large as possible, while more than three years could not be used, because census returns for this purpose are not a reasonably accurate guide for a longer period. But even using three years, it was found that the number of deaths in some occupations was so small as to be worthless in such a study, and consequently not all occupations were included, but only those occupations or groups of occupations were included which might be excepted to give results of some value.

The mortality, or death-rate, prevailing in the various occupations of the male section of communities is a matter which has been previously studied on a large scale on at least eight occasions. Of them one was in Scotland, it was published in the Supplement to the Thirty-eighth Detailed Annual Report of the Registrar-General; four were English studies, and were published in the Supplements to the Twenty-fifth, Thirty-fifth, Forty-fifth, and Fifty-fifth Annual Reports of the Registrar-General of England and Wales; two were Parisian studies—they were published in "Annuaire Statistiques de la Ville de Paris for the years 1889 and 1895"; the eighth was a Swiss study, and was published in a report issued by the Swiss Statistical Bureau dealing with the causes of death during the years 1871 to 1890.

In making these studies, there are two statistical difficulties or sources of error, the one is the difficulty of making reasonable comparison between somewhat dissimilar quantities, the other is the want of numbers sufficiently large to enable rates

to be stated with certainty. The former difficulty is dependent on the varying age distribution in the various occupations, the latter on the limited numbers engaged in the occupations. Methods for overcoming the former difficulty have been devised and used in previous studies, but, so far as I am aware, in previous studies there has been no endeavour to systematically allow for probable error arising from paucity of observations. Before describing the method of tabulation which I have adopted, I shall briefly describe those previously used.

In the Swiss study the method of tabulation used was to arrange the various occupations in order of magnitude of death-rate in each of four age periods, and class those occupations as subject to low mortality which were found to be near the top of such tabular statements, and those occupations as subject to high mortality which were low in such tabular statements. Thus agriculture is found to be fourth in the list in three age periods and second in one age period, and is included among those occupations subject to low mortality; while painters and decorators are found near the foot of the list in all age periods, and are described as subject to high mortality.

In the Parisian studies, or more accurately in a paper read by the author of the Parisian studies at the seventh International Congress of Hygiene and Demography, a graphic method was used. This consisted in the representation of the death-rates of all males in the age periods by a curve in a diagram, while a superimposed curve represented the death-rates of the occupation under consideration, the difference between the two curves showing the difference between the mortality of the occupation and that of all males. This method has the advantage of showing differences at a glance and concisely.

In the English studies and in the previous Scottish study a method of tabulation by means of a comparative mortality figure was used. The comparative mortality figure was arrived at in this manner. The first step was to find the number of all males in the age periods under study which, when subject to the recorded death-rates of all males, would yield exactly 1000 deaths. Having that number, it was then divided proportionally into the numbers in the different age periods, and having those numbers apply to them the death-rates found in the occupations, the calculated number of such deaths

being the comparative mortality figure. Thus in the previous Scottish study it was found that there were 36,201 deaths among 748,965 males of between 25 and 65. From that it was calculated that there would be 1000 deaths among 62,067 males of those ages; and that of the 62,067 persons, 40,208 would be between 25 and 45 years old, and 21,859 between 45 and 65. Applying the death-rates of the two age periods found in the clerical profession to these populations (40,208 and 21,859), it was found that in all 581 deaths would result. That number is the comparative mortality figure, and indicates that were the death-rates among the clerical profession common to all males, there would have been only 581 deaths instead of each 1000 deaths. This method has advantages, the calculations are simple, the difficulties of fair comparsion are overcome, and the results are concise and definite. But this method, like those mentioned above, assumes that the rates found indicate the true death risks, and makes no allowance for probable errors.

The method which I have adopted in this study was suggested to me by Professor Karl Pearson. It consists of ascertaining the amount of probable error, and loading the ascertained rates with those errrors. The probable error per unit is calculated by this formula, $\frac{0.67449}{N}\sqrt{\frac{m.n.}{N}}$, and the amount of variation thus found is expressed with each rate thus—rate ± probable error.[1] Having ascertained the rates and the amount of variation, only those rates are taken as decidedly different whose difference is at least three times the amount of the combined probable errors. Thus the death-rates among ministers, clergymen, and priests in the 25 to 45 age period is found to be 3.88 ± 0.44, that of all males in that age period 9.32 ± 0.05. The difference between the crude rates is 5.44, the amount of three times the probable errors is only 1.47, and therefore the death-rate of the clerical profession in that age period is taken as decidedly less than the corresponding death-rate of all males. On the other hand, the death-rate of fishermen in the same age period is found to be 8.75 ± 0.32; this rate is not taken as decidedly different from the corresponding rate of all males, because the crude

[1] In the appended table the plus or minus probable error is shown by the figures in parentheses.

difference between the crude death-rates, 0.57, is less than three times the amount of the combined probable errors, 1.17.

Unfortunately the use of this new method prevents a simple and comprehensive comparison of results such as is got when comparative mortality figures are used and a new method of comparison is required. The one I have adopted is to divide the occupations according to whether the death-rates in one or both of two age periods are smaller, greater, or not decidedly different from the corresponding death-rates of all males. By this means the occupations are divided into six classes, and if an occupation occurs in a class with low death-rates, it is held to be one with a low mortality, while if it occurs in a class with high death-rates, it is held to be an occupation with high death-rates. This method has not the nicety of that in which comparative mortality figures are used, but it has, I think, the advantage of not overestimating the value of the observations.

The classification adopted is as follows :—

CLASS 1.—Occupations with death-rates less than the corresponding death-rates of all males in both age periods.

CLASS 2.—Occupations with a death-rate in one age period less than that of all males, but in the other age period a death-rate not decidedly different from that of all males.

CLASS 3.—Occupations with death-rates in both age periods not decidedly different from the corresponding death-rates of all males.

CLASS 4.—Occupations with a death-rate lower than that of all males in one age period, while that of the other age period is greater than the corresponding rate of all males.

CLASS 5.—Occupations in which there is a high death-rate in one age period, and in the other a death-rate not decidedly different from that of all males.

CLASS 6.—Occupations in which the death-rates in both age periods are greater than the corresponding death-rates of all males.

Occupations in Class 1 may be taken as those with the lowest mortality, those in Class 6 as those with the highest mortality, those in Classes 3 and 4 as having a mortality little, if any, differing from that of all males, and occupations in Classes 2 and 5 as having a mortality intermediate between the average and the extremes.

A great objection to the introduction of a new method of tabulation is that comparison with previous work is rendered

less simple; but, notwithstanding that objection, I have thought it right to introduce this new method rather than use one which may overestimate the accuracy of the results of the study.

Professions.—Among the professions, four are included among the selected occupations. These are the clerical, including ministers, clergymen, and priests; the legal, including advocates and solicitors; the medical, including physicians, surgeons, and dentists; and the teaching, which includes schoolmasters, teachers, professors, and lecturers. If these four are considered together, they are found to be in Class 1, and to have in both age periods a death-rate decidedly less than those of all males of the same ages; but if considered separately, it is found that the clerical profession is the only one in Class 1, the legal and teaching professions being in Class 2, and the medical in Class 3. The previous Scottish and English studies agree with this result, in that in them it was found that the clerical profession was the healthiest of the four and the medical the least healthy. The results of the continental studies however did not agree, the clerical not being in all cases the healthiest, nor the medical the least healthy.

Farming and allied occupations.—Five such occupations are included among the selected ones. These are farmers and graziers, agricultural labourers and farm servants, gardeners, nurserymen, and seedsmen, shepherds, and gamekeepers. These occupations, when considered both collectively and separately, are found to be in Class 1, to have low death-rates in both age periods, and consequently to be exceptionally healthy occupations. The results of previous studies so far as comparable agree with this result.

Fishermen.—This is the only nautical occupation included among those selected for this study. It is found to be in Class 2, having a death-rate not decidedly different from the average in the earlier age period, but a low death-rate in the older age period. In the previous Scottish and English studies the mortality figure was found to be low.

Horsemen.—Among the selected occupations there are two concerned with the care and use of horses—the one includes coachmen, grooms, and cabmen, the other carters, carriers, and vanmen. Collectively these occupations are in Class 6. Considered singly, the former is found to be in Class 6, having high

death-rates in both age periods, and the latter in Class 5, having a high death-rate over 45, but a death-rate under 45 not decidedly different from the average. Results of previous studies, so far as they are comparable, agree with this.

Commercial travellers.—This occupation is found to be in Class 2, and to have a low death-rate in the younger age period, and in the older period one not decidedly different from the average. In the previous Scottish study the reverse, was found, the younger death-rate was high, while the older one was low. In that study the comparative mortality figure was low. In the last two English studies the mortality figure was low. In the Paris study the death-rate in the earlier period was low, and that in the older period high.

Liquor trade.—Among the selected occupations there are three concerned with the liquor trade. These are—(1) Malsters, brewers, and distillers; (2) hotelkeepers, publicans, and wine and spirit merchants; and (3) barmen and cellarmen. When considered collectively, these occupations are found to be in Class 6, and to have high death-rates in both age periods; but when considered separately, it is found that the first, malsters, etc., are in Class 3, with death-rates not decidedly different from the average, while the other two, hotelkeepers, etc., and barmen, etc., are both in Class 6.

Domestic service.—There was only one occupation of this nature among those selected; it includes hotel and other domestic servants (male only; female occupations were not studied), and it was found to be in Class 3, with death-rates in both age periods not decidedly different from the average.

Clerks.—Three occupations of this nature were included among those selected. These are law clerks, commercial and railway clerks, and bank and insurance service. These occupations collectively are found to be in Class 3, with death-rates not decidedly different from the average; but when considered separately, law clerks are found to be in Class 3, commercial and railway clerks in Class 5, their death-rate in the 25 to 45 age period being higher than that of all males; while bank and insurance service is found to be in Class 2, having a low death-rate in the 45 to 65 age period. In previous studies, law clerks were found to have high comparative morality figures, a result not corroborated in this study. Owing to differences of selections of occupations, the results of previous studies

regarding commercial and banking and insurance clerks are not readily available for comparison.

Chemists.—Among the selected occupations there is one which includes chemists, druggists, and manufacturing chemists, and it is found to be in Class 6, and to have high death-rates in both age periods. Owing to differences of classification of occupation, this result cannot be fairly compared with the results of previous studies.

Shopkeepers.—Five of the selected occupations are of this nature, grocers and provision merchants, drapers, linen-drapers and mercers, ironmongers and metal-dealers, coal merchants, and general shopkeepers. When the returns of the five are massed and considered collectively, it is found that the occupation of shopmen is healthy, for it is found that the death-rates in both age periods are low, and the group of occupations is in Class 1. When considered singly, grocers are found to be in Class 2, drapers in Class 1, ironmongers in Class 1, coal merchants in Class 3, and general shopkeepers in Class 2. The results of previous studies are similar, for in the Scottish and in each of the English these occupations were found to have low mortality figures.

Preparation and sale of food.—Five of the selected occupations belong to this group. They are dairymen and milk-sellers, fishmongers, poulterers and game-dealers, butchers and meat salesmen, millers, and bakers, confectioners, and biscuit makers. Collectively, this group of occupations is found to be in Class 5, having a death-rate in the 25 to 45 age period not decidedly different from the average, but one in the 45 to 65 age period higher than the average. Considered separately, dairymen are found to be in Class 3, fishmongers in Class 3, butchers in Class 3, millers in Class 2, with a low death-rate in the younger age period, and bakers in Class 4, with a low death-rate in the younger period, but a high one in the older period. The results of previous studies, so far as available, generally agree with those results, except those regarding butchers, for in each of the English studies, and in the previous Scottish study, that occupation was found to have a high mortality figure, and high death-rates among butchers were also found in the Swiss study, in the Parisian studies, however, no excessive death-rates among butchers were found.

Hairdressers.—This occupation is found to be in Class 6,

and to have high death-rates in both age periods, results which agree with those of all previous studies.

Tailors and clothiers.—This occupation is also found to be in Class 6, with high death-rates in both age periods. High mortality figures for this occupation were found in the previous Scottish study and in the second and third English studies, and high death-rates were found in the Swiss study, but a low mortality figure was found in the fourth English study, and low death-rates in the Parisian studies. These results are somewhat contradictory.

Leather workers.—Three occupations of this nature are among the selected ones. These are boot and shoe makers and dealers, skinners, tanners, and curriers, and saddlers and harness and whip makers. Collectively, these three occupations are found to be in Class 3, with death-rates in both age periods not decidedly different from the average. Individually, skinners, etc., and tanners, etc., are also found to be in Class 3 ; but bootmakers, etc., are found to be in Class 5, as the rate among them in the older period is high. In the previous Scottish study, bootmakers were found to have a high mortality figure, while skinners and saddlers had a low one. In the English studies, bootmakers had a low mortality figure, but saddlers a high one in one instance and a low in two instances. In the Parisian studies, bootmakers and saddlers were found to have high death-rates, while skinners had low death-rates. In the Swiss study, saddlers had high death-rates, and bootmakers a low one in the younger age period and a high one in the older age period. The results of this and previous studies are thus not altogether in agreement.

Printers.—The occupation of printer and lithographer is found to be in Class 3, a result in general agreement with those of the last two English studies and the previous Scottish study, but in disagreement with the result of the first Parisian study, when this occupation was found to have high death-rates in both age periods.

Watchmakers.—The occupation of watchmakers and jewellers is also found to be in Class 3, a result closely agreeing with those of the last English and the previous Scottish studies.

Paper manufacture.—The occupation of papermaker is found to be one with low death-rates in both age periods, and to be

in Class 1. This agrees with the results of the previous Scottish study and those of the English studies.

Brick, cement, pottery, and glass manufacture.—This group of occupations is found to be in Class 3, and to have in both age periods a death-rate not decidedly different from the average. Results of previous studies are not available for purposes of comparison.

Textile manufacture.—There are four of the selected occupations which are of this nature, cotton flax and linen manufacture, wool and worsted manufacture, textile-bleachers printers dyers and calenderers, and jute manufacturers. These four together are found to be in Class 1, with death-rates in both age periods less than the average. Separately, the cotton, wool, and jute manufacturers are also found to be in Class 1, while the bleachers and dyers are in Class 4, with a low mortality in the younger age period, and a high mortality in the older age period. The results of previous studies regarding cotton manufactures are contradictory, for in the previous Scottish study the mortality figure was found to be slightly low, while in the last two English studies it was somewhat high. Woollen manufacture in the last Scottish study was found to have a very low mortality figure ; in the last two English studies the mortality figure was very little different from the average. Bleachers and dyers were found in the previous Scottish study to have a high mortality figure, as was also the case in each of the English studies, but in the Swiss study the death-rates of that occupation were found to be low. Jute manufacture in the previous Scottish study was found to have a high mortality figure. These results are in several instances contradictory. Excluding the bleachers and dyers from the group, it is found that these textile manufactures are subject to a decidedly low death-rate in both age periods.

Building trades.—Eight of these occupations are among those selected. These are builders, masons and bricklayers, slaters and tilers, plasterers, plumbers and gasfitters, painters, paperhangers and glaziers, cabinetmakers, French polishers and upholsterers, carpenters and joiners, and sawyers. When considered collectively, these occupations are found to be in Class 3, and to have death-rates in both age periods closely approximating to those of all males. When considered separately, it is found that carpenters and joiners are in Class 1, with low

death-rates in both age periods; plumbers and gasfitters and sawyers are both in Class 2, with a low death-rate in the younger age periods, and death-rates not decidedly different from the average in the older age period; slaters and tilers, plasterers, cabinetmakers, French polishers and upholsterers are each in Class 3, with death-rates in both age periods not decidedly different from the average; painters, paperhangers and glaziers are in Class 5, with a high death-rate in the older age period, and one not decidedly different from the average in the younger age period; while builders, masons and bricklayers are in Class 6, with high death-rates in both age periods Builders, etc., in the last Scottish study were found to have a high mortality figure, but the results in the English, Parisian, and Swiss studies did not show that result uniformly. Slaters and tilers have, with the exception of the English 1881 study, in each previous study been found to be subject to high death-rates. In the previous Scottish study plasterers were found to have death-rates little different from the average. Plumbers and glaziers were in the previous Scottish study found to have a low mortality figure. Painters in those previous studies which are comparable to this were found to have high death-rates. Cabinetmakers, etc., carpenters, etc., and sawyers have in previous studies been found to have low mortality figures and low death-rates. Thus regarding the building trades the results of the present study generally agree with those of the previous studies.

Coopers.—The occupation of cooper is found to be in Class 2, with a low death-rate in the younger age period, and a death-rate not decidedly different from the average in the older age period. In the previous Scottish study a very similar result was found. In the Parisian and Swiss studies this occupation was found to have high death-rates.

Cycle, motor, carriage and waggon makers.—This occupation is also found to be in Class 2, with a low death-rate in the younger period, and one not decidedly different from the average in the older period. Very similar results were found in the previous Scottish and English studies.

Shipwrights, ship and boat builders.—This occupation is found to be in Class 1, and to have in both age periods death-rates decidedly less than those of all males. In the previous Scottish and in the English studies similar results were found.

Iron manufacture.—Five of the selected occupations are connected with iron manufacture. These are, iron and steel manufacturers and ironfounders, blacksmiths and strikers, engine and machine makers, boilermakers, erectors fitters and turners. Considered collectively, these occupations are found to be in Class 5, with a death-rate under 45 not decidedly different from that of all males, and a death-rate over 45 greater than that of all males. When considered separately, it is found that erectors and fitters are in Class 1, with low death-rates in both age periods ; blacksmiths and strikers are in Class 2, with a low death-rate under 45, and an average one over 45 ; engine and machine makers, and iron and steel manufacturers and iron-founders are both in Class 5, with an average death-rate in the younger age period, and a high one in the older age period ; while boilermakers are found to be in Class 6, with high death-rates in both age periods. Previous studies, so far as comparable, are somewhat contradictory ; for instance, in the last English study, ironfounders were found to have the highest mortality of this group, while boilermakers had a mortality figure of less than the average amount. In the previous Scottish study boilermakers were found to have the highest mortality figure of the group, while engine and machine makers had the lowest.

Tinplate goods makers.—This occupation is found to be in Class 3, and to have death-rates in both age periods not decidedly different from those of all males, a result generally corroborated by previous studies.

Brassfounders, finishers, workers.—This occupation is also found to be in Class 3, with average death-rates in both age periods. With that result the previous Scottish study agrees.

Miners and quarriers.—Three of the selected occupations belong to this group ; they are coal and shale miners, ironstone miners, and stone quarriers and dressers. When considered collectively, these occupations are found to be in Class 4, with a low death-rate under 45, and a high death-rate over 45. When considered separately, it is found that both coal and shale miners and ironstone miners are in Class 4, with a low death-rate under 45, and a high death-rate over 45 ; while stone quarriers and dressers are in Class 3, with death-rates in both age periods not decidedly different from those of all males. The results of previous studies are contradictory. In

the last Scottish study, quarriers were found to be the healthiest of the group, and ironstone miners decidedly the least healthy, while coal and shale miners were intermediate. In the English studies, ironstone miners were found to have decidedly small comparative mortality figures and quarriers high ones.

Engine-drivers, stokers, firemen (not railway, marine or agricultural).—This occupation is found to be in Class 2, with a low death-rate under 45, and one not decidedly different from that of all males in the older age period. In the previous Scottish and in the last English studies this occupation was found to have low comparative mortality figures.

Railway service (except clerks).—Railway service is found to be an occupation in Class 1, and to have in both age periods death-rates decidedly less than the corresponding rates of all males. A similar result was found in the previous Scottish study.

Messengers, porters, watchmen.—These occupations are found to be in Class 6, and to have death-rates in both age periods greater than those of all males. Similar results were found in the previous studies.

Hawkers, street-sellers.—These occupations are also found to be in Class 6, with high death-rates in both age periods. In the previous Scottish study and in each of the English studies high mortality figures were also found.

Dock labourers.—This occupation is found to be in Class 3, with death-rates in both age periods not decidedly different from those of all males. In the previous Scottish study this occupation was found to have a high comparative mortality figure; the results of the English studies are not available for purposes of comparison.

General labourers.—This occupation in this as in previous studies is found to be subject to high death-rates, and therefore in Class 6.

I append a tabular statement showing the selected occupations arranged in classes, the death-rates with their probable errors of each of those occupations, and an indication of those occupations in which certain selected causes of death are found to be decidedly more or decidedly less prevalent.

TABLE showing Occupations arranged in Classes according to amount of Death-Rates, and also showing in which Occupations decidedly high or low Death-Rates from certain causes of death are found.

Rates against each occupation show death-rates per thousand in 25 to 45 and 45 to 65 age periods.

\+ indicates death-rate is greater than that of all males.

– indicates that death-rate is less than that of all males.

o indicates that death-rate is not decidedly different from that of all males.

Figures against each class indicate death-rates in two age periods studied, namely, 25 to 45, and 45 to 65. The figures in parentheses indicate the plus or minus possible error.

Occupation.	Tubercle.	Malignant Disease.	Pneumonia.	Bronchitis.	Violence.
All males, 9.32 (0.05), 26.82 (0.11)	...	...	...	...	...
CLASS 1 (–.–).					
Ministers, clergymen, priests, 3.88 (0.44), 17.03 (1.08)	–	...	–	...	–
Farmers, graziers, 4.99 (0.20), 15.89 (0.33)	–	+	–	–	–
Agricultural labourers, farm servants, 4.64 (0.17), 16.48 (0.48)	–	...	–	–	–
Gardeners, nurserymen, seedsmen, florists, 6.07 (0.36), 18.35 (0.70)	–	...	–	...	...
Shepherds, 6.48 (0.50), 13.61 (0.86)	–	...	–	–	–
Gamekeepers, 3.34 (0.40), 14.26 (1.18)	–	...	–	–	–
Drapers, linen drapers, mercers, 7.50 (0.49), 21.09 (1.23)	...	...	–	...	–
Paper manufacture, 5.27 (0.64), 20.03 (1.67)	...	...	–	...	...
Cotton, flax, and linen manufacture, 6.61 (0.61), 22.70 (1.29)	...	...	...	+	–
Wool and worsted manufacture, 3.40 (0.39), 17.21 (1.07)	–	...	–	...	–
Jute manufacture, 6.35 (0.56), 18.14 (1.31)	–	...	...	...	–
Carpenters, joiners, 7.62 (0.19), 21.03 (0.58)	...	...	...	–	–
Shipwrights, ship and boat builders, 7.36 (0.27), 21.54 (0.68)	–	...	–	...	...
Erectors, fitters, turners (engine and machine), 8.08 (0.33), 21.57 (0.84)	–	...	...	–	–
Railway service (except clerks), 8.43 (0.28), 22.04 (0.67)	–	–	...	–	+
CLASS 2 (o. – or – .o).					
Fishermen, 8.75 (0.32), 17.23 (0.61)	...	...	–	–	+
Bank and insurance service, 8.47 (0.52), 21.99 (1.22)	...	...	–	–	–
Ironmongers, metal merchants, 8.44 (0.81), 19.87 (1.73)	...	...	...	...	...
General shopkeepers, dealers, 12.14 (1.23), 17.32 (1.83)	...	...	...	...	...
Advocates, solicitors, 6.05 (0.61), 25.49 (1.90)	–	...	–	...	–

Occupation.	Tubercle.	Malignant Disease.	Pneumonia.	Bronchitis.	Violence.
Schoolmasters, teachers, professors, lecturers, 5.96 (0.48), 23.19 (1.40)	–	...	–	–	–
Commercial travellers, 7.39 (0.44), 27.07 (1.39)	...	...	...	...	–
Grocers, provision merchants, 7.80 (0.35), 25.73 (1.01)	–	...	–	–	–
Millers, 5.77 (0.83), 30.23 (2.25)	...	...	...	...	...
Plumbers, gas-fitters, 7.69 (0.51), 24.95 (1.54)	...	...	...	–	–
Sawyers, 7.24 (0.64), 23.02 (1.62)	...	...	...	...	...
Coopers, 6.23 (0.64), 27.10 (1.74)	...	...	...	...	–
Carriage, waggon, motor, cycle makers, 7.28 (0.60), 23.71 (1.58)	...	...	–	...	–
Blacksmiths, strikers, 7.56 (0.34), 27.28 (0.80)	...	...	...	+	...
Engine-drivers, stokers, firemen (not railway, marine, or agricultural), 7.65 (0.36), 24.78 (0.90)	–	...	...	–	+
CLASS 3 (0.0).					
Physicians, surgeons, dentists, 9.66 (0.82), 27.36 (2.16)	...	...	...	...	...
Malsters, brewers, distillers, 7.83 (0.67), 23.83 (1.75)	...	...	...	...	...
Hotel and other domestic servants, 9.88 (0.77), 24.12 (2.27)	...	...	...	+	...
Law clerks, 8.57 (0.85), 26.63 (3.41)	...	...	...	...	...
Coal merchants, 9.56 (0.98), 27.64 (1.86)	...	...	...	...	...
Dairymen, milksellers, 8.36 (0.98), 23.51 (1.91)	...	...	...	...	...
Fishmongers, poulterers, game-dealers, 11.59 (1.28), 29.99 (2.76)	...	...	...	...	–
Butchers, meat salesmen, 10.22 (0.57), 26.64 (1.58)	...	...	...	–	–
Skinners, tanners, curriers, 7.24 (0.82), 24.93 (2.83)	...	...	...	...	...
Saddlers, harness and whip makers, 8.06 (1.18), 27.32 (2.87)	...	...	...	. .	...
Printers, lithographers, 10.52 (0.55), 31.58 (1.59)	+	...	...	–	–
Watchmakers, jewellers, 9.65 (0.90), 23.53 (2.02)	...	...	...	...	...
Brick, cement, pottery, and glass manufacture, 9.27 (0.62), 29.87 (1.65)	...	...	+	...	...
Slaters, tilers, 9.81 (0.75), 25.00 (1.56)	...	...	...	...	...
Plasterers, 9.86 (0.78), 22.36 (1.72)	...	...	...	...	...
Cabinet-makers, French polishers, upholsterers, 8.88 (0.57), 25.13 (1.33)	...	...	...	...	–
Tinplate goods makers, 8.77 (0.88), 27.86 (2.41)	...	...	...	...	...
Brass-founders, finishers, workers, 9.34 (0.78), 24.14 (1.82)	...	...	...	...	–
Stone quarriers and dressers, 8.45 (0.45), 25.02 (1.08)	+	...	...	...	+
Dock labourers, 10.93 (0.57), 28.83 (1.22)	...	...	+	...	+
CLASS 4 (+.– or –.+).					
Bakers, confectioners, biscuit makers, 7.99 (0.37), 32.12 (1.14)	...	...	...	...	–
Coal and shale miners, 7.13 (0.15), 30.22 (0.53)	–	–	...	...	+
Ironstone miners, 6.47 (0.93), 41.16 (3.48)	–	...	...	+	...
CLASS 5 (+.0 or 0.+).					
Carters, carriers, vanmen, 8.90 (0 27), 29.89 (0.83)	–	...	+	...	+
Commercial clerks (including railway clerks), 10.15 (0.30), 25.84 (0.81)	+	–	...	–	–

Occupation.	Tubercle.	Malignant Disease.	Pneumonia.	Bronchitis.	Violence.
Boot and shoe makers, dealers, 10.71 (0.49), 32.61 (0.93)	+	+	...	+	...
Textile bleachers, printers, dyers, calenderers, 9.89 (0.55), 32.44 (1.34)	...	..	...	...	...
Painters, paperhangers, glaziers, 9.11 (0.45), 32.17 (1.30)	...	...	...	...	...
Iron and steel manufacture, 9.98 (0.26), 35.14 (0.79)	...	...	+	+	...
Engine and machine makers, 10.55 (0.48), 32.29 (1.27)	+	...	...	...	...
CLASS 6 (+.+).					
Coachmen, cabmen, grooms, 12.52 (0.50), 33.19 (1.25)	+	+	+	...	...
Hotel-keepers, publicans, wine and spirit merchants, 17.66 (0.86), 42.31 (1.61)	+	+	+	..	...
Barmen, cellarmen, 18.76 (0.96), 51.83 (4.22)	+	...	+	...	...
Chemists, druggists, manufacturing chemists, 13.83 (1.02), 38.41 (2.10)	...	...	...	...	...
Hairdressers, 16.94 (1.30), 41.01 (4.34)	+	...	...	...	...
Tailors, clothiers, 10.82 (0.40), 32.32 (1.02)	+	...	...	...	—
Builders, masons, bricklayers, 11.47 (0.32), 33.48 (0.62)	+	...	+	+	...
Boiler-makers, 17.39 (0.77), 43.03 (1.80)	+	...	+	+	+
Messengers, porters, watchmen (not Government or railway), 16.30 (1.10), 47.86 (1.77)	+	+	+	+	+
Hawkers, street sellers, 16.59 (1.40), 52.27 (2.54)	+	+	+	+	...
General labourers, 18.05 (0.34), 55.77 (0.75)	+	+	+	+	+

Meeting VII.—May 2nd, 1906

PROFESSOR W. S. GREENFIELD, *Vice-President, in the Chair*

I. ELECTION OF HONORARY MEMBER

Professor P. Ehrlich, Frankfort-on-the-Maine, was elected an Honorary Member of the Society.

II. EXHIBITION OF PATIENTS

1. *Mr Alexis Thomson* exhibited a patient on whom he had performed CHOLECYSTENTEROSTOMY. The patient, a man aged sixty, had suffered from progressive failure of health and painless jaundice of five weeks' duration. The distended gall-bladder

was easily palpated. The diagnosis lay between carcinoma in the region of the bile papilla and a tumour of the head of the pancreas, inflammatory or malignant. Cholecystenterostomy was performed four months ago; the gall-bladder, which contained nearly a pint of dark thick bile, was anastomosed to the hepatic flexure of the colon. No tumour was discovered. After the operation, bile re-appeared in the motions, and the jaundice gradually disappeared.

2. *Mr J. M. Cotterill* exhibited—

(*a*) A CASE OF ANGIOMA OF THE OPHTHALMIC ARTERY, causing marked proptosis of the right eye with considerable loss of vision.

(*b*) A CASE OF RUPTURE OF THE INTERNAL SEMILUNAR CARTILAGE OF THE KNEE-JOINT, successfully operated on. The patient was shown to illustrate a modified vertical line of incision, which Mr Cotterill had recently used, and thought superior to the usual transverse incision.

III. Exhibition of Specimens

Mr J. M. Cotterill exhibited—

(*a*) A LARGE HYDRONEPHROSIS DUE TO CONGENITAL STENOSIS OF THE URETER, successfully removed from a young man.

(*b*) A LARGE HYDATID CYST, removed from a patient from Shetland aged four years.

(*c*) A brain exhibiting a GLIOSARCOMATOUS TUMOUR OF THE FRONTAL LOBE.

IV. Demonstrations

1. *Mr Alexis Thomson* and *Dr Woodward* gave a demonstration of Dr Klapp's system of exercises devised for the treatment of scoliosis.

2. *Mr L. C. Peel Ritchie* demonstrated—

(*a*) A PORTABLE RHEOSTAT for use with Kirstein's lamp for endoscopic examination with laryngeal autoscope, œsophagoscope, bronchoscope, urethroscope, etc., and for direct attachment to cystoscope, etc.; special mirror for lamp for demonstration purposes.

The rheostat weighs 3 kilos., and reduces from 230 to from

9 to 3 volts, and along with Kirstein's lamp provides the most convenient apparatus for all endoscopic examinations. The lamp permits of focussing the rays at very varying distances, making it equally suitable for use with the long œsophagoscopic or short urethroscopic tubes. A special large mirror attachment and adjustable holder were shown which serve for demonstration of the illuminated object to the bystanders.

(*b*) NEW LIGATURE WINDERS. These consist of an aluminium frame with a couple of adjustable notches to hold the ends of the ligature and obviate the use of knots. One side of the winder is rounded to form a handle. Being made of aluminium the winders are specially suited for use with iodine sterilised catgut. The winders are stored ready for use in a small metal box fitted at the bottom with a wire spiral to hold them in position.

V. ORIGINAL COMMUNICATIONS

1. THE PRACTICAL SIDE OF AN INFANT'S MILK DEPOT

BY W. ROBERTSON, M.D., D.P.H., Medical Officer of Health, Leith

I AM pleased to be able to contribute to the discussions of this Society by bringing before it the subject of Municipal Milk Depots. One usually reads pæans of praise regarding these admirable institutions. I do not intend to follow that precedent, but wish rather to place before you the weaknesses and shortcomings of these factors established for the reduction of a particular form of infantile mortality. I must, however, protect myself by stating that despite my knowledge of these imperfections, I still believe in the value of the municipal milk depot, and shall continue to repose my faith in it until something very much better is suggested and put into practical operation. Beginning at the crux of the whole subject of infant feeding by hand, one is compelled to turn one's attention (the vexed question, I should add) to the present-day milk traffic. Last winter Dr Aitchison Robertson made out a very strong case against the milk-supply of our cities. I do not intend to traverse the same ground so fully and clearly gone over by him. I desire rather to attack the problem in order to anticipate the argument advanced against the

milk depot principle. It has been said, and is still said, that a milk depot is merely a clumsy cloak for an impure milk-supply. In other words, that the only logical and practical step toward the end in view—to wit the provision of wholesome food for infants—is to wait until the milk-supply has been established upon an unimpeachable basis.

I can hardly imagine there is any one who will cavil at that ideal; but let us clearly understand what this standard of efficiency demands. My personal experience affords abundant testimony to show how enormous are the obstacles that confront us. In the first place, a pure milk-supply ought to come from cows accustomed, for the greater part of the year at least, to chew the cud upon some meadow or rich pasture land. The cows, the milkers' hands, the dishes and pitchers must also be scrupulously clean; and, to preserve the milk in all weathers, chilling ought to be resorted to. Now that is the ideal; an excellent ideal, to which we must aspire. Consider, however, the condition of affairs in Edinburgh and Leith. In neither district are the cows privileged to pasture, save perhaps in isolated suburban instances. The dairymen are in most cases utterly opposed to move out of the track trodden for years by their forefathers. They will provide good cowsheds, and will make hurried attempts to keep their cows clean; but if they begin to find fault with their servants the latter will not only resent interference, but will without compunction leave the dairyman in the lurch. Time forbids their putting into operation what they are pleased to term fads of health officials. During the summer months they prefer to add formalin or boracic acid to milk rather than take time to pass the new milk over a chiller. The utmost vigilance is required to keep down adulteration by the addition of water, skimmed milk, and preservatives. And to complete the farce, glaring cases of adulteration—almost self-confessed adulteration—are taken before the Sheriff, who shelters himself behind an absurdly worded regulation issued by the Board of Agriculture, and dismisses cases without number. Indeed, so many rebuffs have been experienced in Edinburgh and Midlothian that few cases are taken before the Court. In Leith we select our cases, because we know that in only very glaring instances have we any hope of success. It is passing strange that in Glasgow the least aberration from the milk standard is met by

a good fine. Obviously, then, in the East of Scotland adulteration is encouraged. It has developed into a fine art. Every dairyman in Midlothian will frankly tell you that it is easy to secure milk well above the standard, but, figuratively speaking, he winks his eye, since he knows exactly how much water he may add and yet be safe from detection or conviction. It is lamentable when one has to confess that it is necessary on the part of the official to watch some dairymen as a cat watches a mouse. Last year an outbreak of diphtheria was traced to a dairy in Leith at which several cows were discovered to be suffering from a teat eruption. As soon as the connection between the dairy and the disease was established, the dairyman was asked to isolate his affected cows, and on no account to sell their milk for human consumption. This he faithfully promised to do. At the end of a week there was no cessation in the notifications, and as the milk-supply was the same in each invaded house, I began to think a mistake had been made in assigning blame to the teats. Incredible as it may appear, inquiry elicited the fact, through one of my inspectors, that as "the doctor had been talking nonsense," the milk continued to be sold. To attempt to deal with such asinine propensities was pure waste of time. I accordingly stationed relays of inspectors at the cowshed at 5 A.M., 11 A.M., and 4 P.M. every day for a fortnight, it being their duty to insist that every drop of infected milk must be poured down the drains. The destruction of the infected milk checked the epidemic. The dairyman lost money, but to this day believes that I was in error. I shall pass over that experience. It is an instance, and a very striking instance, of what the official has to contend against, even when law and right are on his side, to protect the citizen.

One often hears it said, and I am in entire agreement with the remark, that cowsheds should not be found in towns. Let me clear away any doubts that may be in your minds when I inform you that we have no powers to remove already existing cowsheds from cities, unless the buildings are in a tumble-down condition, or are creating nuisance which is not remediable.

Another practical illustration will add force to that statement. Three years ago the War Office authorities communicated with me regarding a proposal to erect a large cowshed near the drill ground of Leith Fort. Sympathising with their

effort, I tried to do what lay in my power to prevent what would undoubtedy develop into a nuisance; but I was met with the reply that I could not anticipate a nuisance. I could condemn the cowshed when it did actually become a nuisance. Such a procedure, directed against a place costing nearly £2000 to build, would have branded an official as a fit subject for observation.

There is very little excuse for the dairyman. He has his technical journals, which tell him as plainly as they can tell any man that to conduct a milk business with satisfaction to himself and his customers he must be cleanly in every direction. The dairyman of the present day knows perfectly well how important it is to groom cows, and to have clean hands to milk the animals. He knows that cooled milk will keep sweet in the warmest weather, and he knows that up-to-date dairymen do sell milk in clean bottles.

What, then, does all this teach us? I submit that the lesson to be learned is, that as soon as the public is taught to insist upon chilled milk, delivered in bottles, the dairymen will pull up their sleeves and set to work to meet the demand. It is a mistaken idea that a fancifully planned and elaborately built cowshed is a necessary corrolary to a pure milk supply. One naturally prefers to look upon a roomy and costly cowshed, but the most advanced dairyman in England at the present time boasts that, while his cows are housed in wooden sheds, he sells their product in as pure a condition as it is possible to collect it. The motive power is the intelligence of the dairyman himself. But beyond all that must be the momentum of public clamour, which will in time impel the sluggard to move with those of his fellow-men who have displayed enterprise and reap the benefit.

The medical officer of health can only administer laws and make suggestions. It rests with the medical practitioner to teach his patients that what is best for them and their offspring is a milk-supply collected under what I might term aseptic precautions. If, then, we are to obviate the necessity for milk depots, we must do away with that necessity. I am perfectly convinced that a persistent clamour for pure milk on the part of the public will soon be practically answered. In Leith we have a case in point. I promised to support a dairyman if he would help me. Each of us has adhered to his bargain, and

he has had no reason to regret the extra trouble he undertook to have clean hands, clean cows, and to filter and chill his milk, which is dispensed in bottles to a good few families.

Depend upon it, if the dairyman woke up one fine day to realise that his customers refused to be served from a pitcher the lid of which had been removed over and over again in dusty thoroughfares and diverse places, we should soon discover a general tidying up all round amongst the milk-selling fraternity. I repeat again, as long as our citizens are pleased to take what is offered to them without demur, then so long must our infants labour under the deleterious influences of an unsatisfactory milk supply.

It is to counteract gross pollution on the one hand, and ignorant methods of feeding on the other, that the present-day milk depot has been established. The long septic feeding-tube is done away with, methodical feeding is taught, and mothers in general are afforded an object-lesson in correct principles of infant feeding.

Unfortunately, the comprehensive term "infantile mortality" has come to be most loosely applied in relation to the milk depot. Many enthusiasts, without giving the subject the serious consideration which it deserves, have jumped to the conclusion that with the institution of a milk depot there must come an immediate reduction in the infantile mortality of a district. A moment's reflection will show that measles and whooping-cough may be controlled by compulsory notification, and that the provision of prepared milk will not avert the deaths of infants from overlying, tuberculous meningitis, and other conditions that claim many victims before they reach the age of one year. But when investigations inform us that every year many infants are sacrificed at the altar of ignorance, we look for causes, and discover that gross neglect in feeding is the prime factor in bringing about so many diarrhœa deaths. Judicious breast-feeding I shall merely mention. Its importance cannot be overestimated. The present-day mother, be she rich or poor, surrounded as she is by the bewildering hum of gaiety and cheap amusement, seeks at the earliest moment to be relieved of a tie. The feeding-bottle is a ready refuge. The milk depot has to come to the rescue. Incidentally I must mention that, as accessory to the milk depot, first place must be given to a lady health visitor. Popping out and in as she does at

unexpected moments upon those whom she picks out as requiring observation, her work is of great value. It is educative, and it must be a very abstruse mind indeed that cannot grasp the fact that all the trouble taken by the "sanitary lady," as she is called, must be for the good of the child being jealously observed. Not a few infants have been snatched from the inevitable result of ignorant guidance by the persistent efforts of our tactful and kindly lady inspector, Miss Armour.

To come to the depot itself, let me briefly outline its methods. In the first place, the milk-supply was a difficult question. Two summers ago an estimation of six samples of milk, taken from six dairies, demonstrated the fact that the bacterial pollution varied in them between 20,000 and 250,000 per c.c. To prove what could be done by drawing milk from clean teats with clean hands into a sterile bottle, the bacterial contamination was shown to be only 700 per c.c. Generally speaking, one has to be content with an impurity of at least 12,000 bacteria in a c.c. That, in fact, is looked upon as a standard supply of clean milk. My choice finally rested upon a dairy farm in Haddington, where the cows were for the greater part of the year fed upon natural pasture, and where every precaution was adopted to ensure a clean supply. I have had no reason to regret my choice, the bacterial examination revealing 15,000 bacteria per c.c. After the milk reaches the depot, water, cream, and sugar are added, the proportions of cream and water added varying according to the ages of the children to be fed. It has required much experimenting to arrive at a fairly uniform mixture. The proteids were usually in excess, and the fat low. As a rule, the fat varies between 3 per cent. and 3.4 per cent. After the bottles have been filled with their proper quantities, they are placed in the steriliser, and subjected to the influence of steam at a temperature or 180° F. for twenty minutes. Subsequent to this, to complete the process of Pasteurisation, the bottles are placed in a tank containing cold water. When the depot was first opened, and during the first year and a half of its operations, an attempt was made to sterilise the milk. I use the word "attempt" intentionally, as the context will presently show. The milk was subjected to a temperature of 212° F. for half an hour, a preliminary half-hour being necessary to reach that degree of heat. This procedure was unsatisfactory, first, because the prolonged

heating frequently caramelised the milk; secondly, because the milk was not sterile; and thirdly, because two cases of scurvy were brought to my knowledge, both infants having been exclusively fed upon the prepared milk. Dr Burn Murdoch had one of these cases under treatment at the Sick Children's Hospital, Dr Murray Wood of Leith the other. These two cases, occurring though they did at widely separated intervals, seriously affected the depot, and some time elapsed ere we recovered credit in the eyes of prospective and actual users of the milk. But I must ask you to suspend judgment regarding the evil influence of cooked milk, since at the first note of alarm a careful analysis revealed a lamentable deficiency in fat. This defect was immediately remedied. Pasteurisation and the addition of fat had not been followed by any suggestions of scurvy. It is very significant that some months before the occurrence of our first case, Dr Ashby[1] had reported a case of scurvy in a child fed upon milk prepared at the depot from which I had secured my original recipe.

Another point of very great interest centres round the question of sterility, as applied to milk prepared at a municipal depot. My assistant, Dr Mair, undertook an exhaustive bacteriological examination of 100 bottles of milk believed to be sterile. Dr Mair and I detailed our findings in a paper.[1] His examination was conducted between the months of November and January, at a time when the proliferation of micro-organisms in milk is inhibited by the prevailing temperature. Only 15 per cent. of the bottles were found to contain sterile milk. I am again glad to be able to record my high appreciation of Dr Mair's work. His results, like many side-issues in science, have little heed paid to them, though their importance cannot be overestimated when, as in this instance, their teaching is practically applied. Professor Flügge of Breslau, Weber, and Lubbert had already worked on these lines, and their results afforded us very valuable assistance. Their conclusions were that such a thing as really sterile milk was extremely difficult to secure. With that knowledge in his mind, Flügge suggested that all so-called sterilised milk should be called "heated milk, not sterile" (*Erhitzte Milch, nicht Keimfrei*). A brief review of the

[1] *Brit. Med. Journ.*, London.

bacteriology of milk will indicate wherein lies the difficulty to secure its sterility.

Roughly speaking, there are three groups of organisms found in cows' milk.

First, those belonging to the *Coli*, *Proteus*, and Lactic Acid group. These are normal inhabitants of the mammary ducts of the cow, and can easily be killed by short exposures to heat.

Secondly, the butyric acid bacilli, anærobic, spore-bearing organisms, capable of rapidly decomposing milk with coagulation and gas formation. Their spores can be killed after exposures of an hour to an hour and a half at 212° F. It is interesting to know that Klein's *Bacillus enteritidis sporogenes* belongs to this group.

Thirdly, there is the *Subtilis* group, which Flügge has named the peptonising organisms, since they are capable of decomposing milk with faintly acid or unchanged reaction. It is to these organisms, in fact, that he has called serious attention as being the prime factors in the causation of summer diarrhœa. So resistant are the spores that exposures for two hours at boiling temperature are necessary to kill them.

The point of practical importance to the physician is that these very resistant organisms can exert their evil influence on milk without altering its taste. Another point of moment is that these organisms do multiply in milk maintained at such high temperatures as 80° F. From this it will be realised how unsafe to the infant it is to store its milk in rooms indicating temperatnres of 70° F. or upwards. Such temperatures during summer and autumn do, in point of fact, exist in many houses and kitchens.

I still am at a loss to account for the cause of the scurvy in the two cases mentioned. Toxicity of the milk was a possible factor, taking into consideration the fact that our sterilisation merely invited the development of micro-organisms capable of giving off toxins. Over-cooking was a possible cause, and, lastly, so was fat deficiency. Dr Still had something very trite to say on this aspect of deficiency of fat, or mal-assimilation of it by the infant in relation to scurvy. I refer to his article.[1] The great advantage of Pasteurisation is that it scarcely alters the taste of the treated milk. I have shown that so-called sterilisa-

[1] *Practitioner*, London, 1905, October.

tion is incapable of destroying the peptonising spores. When we prolong the application of a boiling heat to milk it is caramelised.

Experience teaches us that Pasteurised milk carries with it the disadvantage that the cooking, be it only partial, throws the onus of digestion upon the intestines. But if sterilisation, as it has come to be applied to milk, is a wrong procedure, Pasteurisation errs on the safe side by inflicting as little damage as possible on the milk. The authorities in the city of Rochester experienced the difficulties of sterilisation and resorted to Pasteurisation ; but they have advanced a stage further and now supply milk collected at the farm, the milk bottles being immediately packed in ice to restrain bacterial growth. Forty-three daily samples of milk so prepared were analysed, and found to contain an average of 14,000 bacteria per c.c. During the same period, milk as retailed in the city milk-shops yielded an average of 235,000 bacteria per c.c. The valuable feature of the Rochester system is that the milk is bottled almost as soon as it leaves the cows. It passes through no middleman's hands, nor is it buffeted about stations, carts, and stuffy railway waggons. The milk is not prepared for infants at Rochester. To my mind, its great advantage is that its delivery in bottles enables the purchaser to secure very pure milk which, by the exercise of a very little care, can be kept pure by immersing the bottles in a dish of cold water.

This system of providing pure milk is the ideal upon which we must devotedly concentrate our energy and attention.

A serious defect of the British Infants' Milk Depot is that supervision of the children fed upon the milk is very perfunctory. In Leith and in other municipalities where the system is in operation, lady health visitors periodically visit houses where infants are being fed on the milk. In Glasgow, a lady practitioner has been appointed to devote her undivided attention to this work ; but even that step is insufficient when we take into consideration that between 200 and 300 babies are being daily fed on prepared milk in that city. Nothing can equal the system followed in France, where infants are brought to a dispensary to be examined, weighed, and their parents advised as to future management. Weighing is of great moment and value, yet, in this country, mothers abolutely refuse to have their children put in the scales. It is believed to bring bad

luck. Pasteurised milk does not agree with all infants. We attempt to make that clear to parents, but, unfortunately, they lose sight of our hints, and when, as occasionally happens, a child being fed upon the milk fails to progress as the mother would like to see it grow, a regular tirade is preached at the depot milk. The fiery cross goes forth, and from within the radius reached by the aggrieved mother's tongue no infants come to the depot. Bottles are frequently found by the lady inspector inside ovens in which the milk is being prepared for the next meal. One cannot be surprised, therefore, when an indignant mother comes to us with a curdled feed, forgetting in her ignorance that she wilfully invited coagulation and gas formation by the use of her domestic incubator. For young infants each meal is so small that our system is decidedly unpopular. The meagre quantities are viewed with head-shakings. The popular quantities are those found in bottles prepared for babies of two months of age and upwards. Over and over again we detect mothers supplementing the food provided in the bottles.

The vast majority of infants brought to the milk depot are put on the prepared milk because every other form of feeding has been tried without success. And though it can be said that Pasteurised milk disagrees with some infants, we can with confidence assert that cows' milk, as mixed in the home, is the prime factor in compelling mothers to seek relief afforded by Pasteurised milk. As I have said, it may throw the onus of digestion upon the intestines, but many an infant with pinched cheeks, sunken eyes, and wasted frame has been brought back to life and vigour by the timely prescription by the physician of Pasteurised milk. All this means that the depot is called upon to pass a severe ordeal. It has come to be looked upon, too often, as a last resort. That it does come out of the ordeal with credit says much for the principle of preparing milk in suitable quantities and in definite proportions.

As an educational factor, the Leith Depot has been of immense value. The sale of feeding-bottles with long rubber attachments has markedly declined. Many now Pasteurise their milk at home. Those who follow the rules and suggestions given at the depot have nothing but praise for the system.

It must be confessed that we do not always reach the infants who really stand in need of our depot milk. When one

comes to deal with facts, there is no need to beat about the bush. The real truth is that the parents of many slum children pay most attention to the public-house bottle. Infants are allowed to battle for existence upon anything that costs little. To study the life-history of an unfortunate slum infant is to appreciate the philosophy underlying the survival of the fittest. The parents of these children will not take the trouble to come or even send for the milk, but they will cheerfully go to the other end of the town if they learn that whisky is on tap in a friend's house. I hold these people in very poor esteem indeed, and I am strongly impressed with the need for legislation that will enable us to tax careless and dissolute parents, such tax taking the form of a weekly contribution towards the proper feeding of children.

In conclusion, I can say with confidence that our depot has done a vast amount of good. It has saved not a few lives, and has taught many parents how their infants can be rationally reared on the bottle. Among the infants fed upon the depot milk in 1904 there were no diarrhœa deaths; among the other infants born but hand-fed there were thirty-six such deaths. In 1905, three infants fed upon the depot milk died as the result of diarrhœa; of the infants born and not fed on the milk forty-nine died as the result of diarrhœa—in every case these latter were hand-fed babies. It is necessary in the interest of the depot to explain that the water famine in the summer of 1905 affected the farm from which our milk-supply was drawn. The consequence of this was that our milk was not chilled before leaving the farm, and bacterial prolification was permitted to have full sway, our treatment of the milk at the depot being powerless to kill those very resistant spores, which were the means of sowing the seeds of diarrhœa.

2. INGUINAL HERNIA IN THE FEMALE

By E. Scott Carmichael, M.B., C.M., F.R.C.S.Ed.

Although inguinal hernia is more common in the male sex, and the femoral variety in the female, one is apt to overlook the fact that the former is of very common occurrence also in women.

Statistics show that in adults this form of hernia occurs in the female in from 5 to 10 per cent. of all cases. The per-

centage in children of female to male inguinal herniæ would seem to be even higher. In 184 radical cures performed by the author in children in this variety of hernia, 23 were females; while of a total number of 729 radical operations in both sexes treated in the hospital, 76 were in little girls, giving a percentage of 10.

I am indebted to Mr Stiles for permission to make use of his cases, in addition to my own.

The relative frequency of this hernia does not seem to vary with age in the two sexes to any great extent.

For the sake of comparison, however, in children under puberty, the cases occurred at the following ages :—

21 were under 1 year of age.
21 were under 5 years of age.
34 were over 5 years of age.

The ages here represented denote the time at which the radical cure was performed ; and although the history of the date of the appearance of the herniæ is unreliable, it would seem probable that the majority of cases were noticed within the first few years of life, and were probably of congenital origin. Inguinal herniæ in male infants and boys are almost certainly congenital, and one is justified in concluding that this form of hernia in female children is also congenital. The frequency of the congenital forms of herniæ, even up to adult life, would seem to be much greater than was formerly supposed.

As in the male, inguinal hernia is more common on the right side, for of the above cases 39 occurred on the right side, 24 on the left side, and 13 were double.

Contents of the Sac.

Contents were found lying in the sac or were pulled down into the sac in 28 cases, the relative proportions being as follows :—

Fallopian tube and ovary	17
Fallopian tube alone	5
Small intestine	2
Omentum	2
Cæcum and ovary and Fallopian Tube	1
Vermiform appendix and ovary and F. T.	1

In two cases the sac was tuberculous, the clinical examination revealing in each case a nodule, resembling in size and shape the form and character of an ovary or small lymphatic gland.

Hernia of the Ovary and Fallopian Tube.

It is suggestive that the persistence of the so-called canal of Nuck should be associated in such a large proportion of cases—over 30 per cent.—with malposition of the ovary and Fallopian tubes.

An examination of the normal position of these organs in the pelvis of the young child reveals a most striking variation.

In many instances the ovary lies longitudinally alongside the lumbar vertebræ, on the psoas muscle, almost as high up as the lower angle of the kidney; while in others it lies on the false pelvis, in the cavity of the true pelvis, and in some cases just inside the internal abdominal ring.

In how far the descent of the ovary is dependent on the action of the gubernaculum or inguinal ligament is difficult to estimate, but that this band is in some way responsible for the position of the ovary is probable.

According to M'Murrich, the descent of the ovary is due to the slower growth of the inguinal ligament as compared with the abdominal walls, which must lead one to suppose that ovarian hernia is due to a retarded development of this band.

Of 28 cases, in which any content was present in the sac, the ovary and Fallopian tube was present alone or along with another viscus in 24 cases. That the frequency of their presence can be explained by simple mechanical causes is extremely unlikely, and one must rather consider some association with the development of the inguinal ligament as the more probable explanation.

In the truly acquired inguinal herniæ of the adult female the presence of the ovary is then probably a mechanical one. Various authorities, such as Englisch, Bitzako, and others, give a higher percentage of acquired as compared with the congenital form of inguinal ovarian hernia; but its frequency in early childhood, and its close relationship to the canal of Nuck, would lead one to conclude, as in the male sex, the probable congenital origin of many that might otherwise be considered acquired.

Whether the formation of this canal is to be considered analogous to the processus vaginalis of the male, is doubtful.

According to Duplay, Beumier, and others this canal does not exist as a normal protrusion, and although Martin, Waldeyer, and others consider it to occur normally as in the male sex, the consensus of opinion would seem to support the former.

One is inclined, after operation in the above series of cases, to consider that the so-called canal of Nuck is an abnormal protrusion, associated in some way with the development of the inguinal ligament, and caused by the same conditions as lead to a drawing down of the ovary and Fallopian tube. The ovary and Fallopian tube do not lie free in the sac in the same sense as a loop of intestine or omentum, but are intimately associated with its wall, the broad ligament or meso-salpinx springing directly from the posterior aspect of the sac. The length of the canal seems to have a certain bearing on the presence or absence of the ovary, as it is rarely present when this is short, but is nearly always present where the canal is long, and extends well down towards the labium majus.

Lockwood seems to imply that ovarian hernia results from abnormal lengthening of the meso-varium and meso-salpinx, and although in a case recorded by him, in which there was torsion of the ovary, this may have been present, one is unable to support this view from the present series of cases. The attachment of the mesentery to the posterior wall of the sack was frequently short and often markedly sessile, so as to render replacement of the organs of considerable difficulty without first dividing the peritoneal folds. It seems more probable that the frequent presence of the ovary in inguinal hernia is due to the drawing down or protrusion of that portion of the peritoneal covering of the pelvis which gives attachment to the broad ligament, and that this takes place by means of the inguinal ligament.

The relation of the round ligament or lower part of the inguinal ligament to the canal of Nuck is an interesting one. Waldeyer believes that the peritoneum covers the round ligament above and in front, but does not surround it, while Devreux considers it to be attached to the apex of the so-called canal of Nuck.

In the cases operated upon, the round ligament appeared distinct up to the apex of the sac, and then seemed to spread out and become lost on its exterior. Puech believes that these herniæ are due to the traction so exerted by a shortened round ligament.

A large percentage of inguinal herniæ in the female are bilateral, and this tendency seems to be marked the younger the subject.

In babies under 1 year, of the 21 cases 8 were double.
„ „ 5 years, of the 21 cases 2 were double.
over 5 years, of the 34 cases 3 were double.

In adults, on the other hand, the bilateral condition would appear not to be so frequent. In some cases the hernia of the opposite side is not noted till after radical cure has been performed on the one side.

Another point of interest in connection with these herniæ is that the younger the subject, the more often is the ovary and Fallopian likely to be present in the sac. Again referring to the ages,

In 21 under 1 year ovary and Fallopian tube in 13 cases.
In 21 „ 5 years „ „ „ 7 cases.
In 34 over 5 years „ „ „ 2 cases.

It is only fair to state that the ovary and tube were not actually present in the sac at the time of the radical cure in all cases; but in some, by pulling on a fold a peritoneum, the base of infundibuto-pelvic ligament constantly present in the sac, the Fallopian tube, followed by the ovary, came into view.

Lockwood, in a case recorded, refers to the relative position of the ovary and Fallopian tube, in which he describes the former as lying lower than and in front of the tube.

Operation on the present series of cases does not bear out this view, as the tube almost invariably appears first in the sac.

Again, while it is rare to find the ovary alone in the sac without the Fallopian tube, in three of the above cases the tube lay alone in the sac, considerable traction being required to expose the ovary.

In order to replace the ovary and Fallopian tube within the abdomen, it is necessary to divide freely its attachment to the sack, and that brings one to consider whether it is

advisable to remove the ovary, to prevent future trouble, and to what extent herniated ovaries differ from the normal.

In order to satisfy myself on this point, I have examined a large number of children post-mortem to ascertain the normal condition of the ovary, not only as regards its position, but with reference to its general microscopic appearances.

As above referred to, the position of the ovary varies very markedly in different subjects, and presents, in addition, as regards its size and shape, the most striking differences in subjects of the same age, and at different ages.

It may be long, flat, and almost ribbon-like in one instance, while in a child of the same age it may be thick and short, resembling a small bean, while it may vary as to its surface from being smooth and homogeneous to one presenting considerable irregularity from the presence of small follicular cysts. With such evident variation in the normal condition one is chary in removing any herniated ovary in children. I have not yet felt justified in doing so, and therefore cannot give any evidence as to their microscopic appearance.

Birnbaum, in a recent paper, describes the histological appearances of the ovary removed from the hernial sac of an adult. The ovarian stroma would appear to be less dense than normal, and to contain collections of cells taking the form of ducts and bands, and in some cases resembling follicles. These follicles are lined by cylindrical epithelium, the interior often being filled with the same kind of cells. Well formed primordial follicles were not present.

In other cases recorded the ovaries presented the appearance of acute or chronic ovaritis, cystic degeneration, and new growth formation.

Although these changes may be present, many cases are recorded where, in observations in adults, the herniated ovaries functionated, swelling at the menstrual periods. Uterine and ectopic pregnancy have frequently been observed in these cases.

With such clinical evidence of the persistence of ovarian functions, one is not justified in removing such ovaries, at any rate as a routine practice.

In the congenital forms of hernia, there are frequently associated malformations of the other genital organs. Sterility

in the adult is generally present in double ovarian herniæ, while absence of the uterus, uterus bicornis, bipartitus, pseudo-hermaphrodism, and true hermaphrodism, according to Olohausen, are not infrequently present.

Hernia of the Uterus.

The uterus itself may be herniated into the canal of Nuck. I have not observed this in any of the above cases, although in one instance on traction of the ovary and Fallopian tube, the uterine horn appeared at the internal abdominal ring.

Hernia of uterus has been recorded not infrequently. Recent papers by Jophson (1) and Helgenrenier (2) state that it has been recorded in over 40 cases, in 8 of which the uterus was pregnant. It appears to occur most frequently in the adult, in which it seems to be associated frequently with multiple pregnancies, or with hermaphrodism.

The uterus itself presents usually some malformation such as bicornuate conditions. This is most frequent in the congenital forms as seen in children. It is probable that more than half the cases are really congenital, and that in the majority some defect or malformation of the genito-urinary tract is present.

It seems to occur most frequently on the left side, as opposed to the frequency of inguinal hernia in general, which is most common on the right side, and is generally associated with hernia of one or both tubes and ovaries, and small intestine.

Hernia of Bowel and Omentum.

From the frequency with which inguinal hernia occurs on the right side, the cæcum and appendix is a not infrequent content. Although it is probably the most constant portion of bowel found in right-sided herniæ in the males, and more especially in young children (see *British Med. Journal*, Feb. 3rd, 1906), the large sacs seen in these herniæ in the male are rarely seen in the female. In the two cases of the present series the cæcum and appendix were only partially present.

The part of small intestine, when present, is usually, as in the male, the lower part of the ileum, just above the cæcum. This was present in the sac in two cases.

The omentum also was present alone in two cases.

Tuberculosis of the Sac.

Tubercle of the sac of a hernia is not infrequent, occurring in about 3 per cent. of herniæ in young subjects.

It may be entirely unsuspected or may be diagnosed before operation. It generally occurs in one of two forms, first, as a tuberculous nodule, generally towards the apex of the sac. In this form it resembles not infrequently in size and shape the ovary, and except for its somewhat less marked mobility, is difficult to distinguish. It is an isolated nodule of many lining the parietal peritoneum of the abdomen, and is as a rule associated with tuberculous glandular enlargement of the mesenteric glands. It is well in all cases where such an oval swelling is felt in a hernia, to examine carefully the abdominal cavity for evidences of glandular enlargement. The most commonly affected glands are those situated in the lower end of the enteric mesentery and ileo - cæcal junction.

This form of tubercle of the sac is the most favourable, although even in such cases a guarded prognosis must be given.

In the second variety there is a diffuse infiltration of the sac, which is thickened and converted into tuberculous granulation tissue. There is no part of the sac healthy. Examination of the abdomen reveals no abnormality, and there is often little, if any, glandular enlargement.

A recent case operated upon in private practice was that of a child, æt. 18 months, which had been in good health up till shortly before operation. It had been noticed to be out of sorts, not taking its food so well for a week or two, before I saw it, but not sufficiently so to induce the parents to call in a doctor.

The child was well nourished. No thorough examination was made of the abdomen before operation. Radical cure was performed of a right-sided hernia. The sac was markedly tuberculous, presenting the appearances of the second variety. The abdomen was then examined while the child was still under the anæsthetic, but no glandular enlargement, arcites, or distension was present.

A guarded prognosis was given to the parents. The child recovered from the operation, but six weeks later developed tuberculous meningitis, with fatal termination.

The peritoneum of the abdomen in these cases appears wholly converted into a lining of tuberculous granulation. There may be no adhesions and no clinical evidence of its presence until discovered at the time of operation.

Of the two varieties of tuberculosis of the sac, the first is the more common, and can, as a rule, be diagnosed before operation.

Clinical Features and Differential Diagnosis.

Where a swelling is present in the inguinal region in a female subject, there are several possibilities that occur to one in making a diagnosis. When a hernial sac contains bowel, as a rule the diagnosis is easy. The characteristic symptoms of a hernia of the intestine are present.

A condition which is not infrequently met with is the history of slight swelling in one inguinal region. On palpation an oval swelling, the size of a bean, is felt. Pressure in the adult will usually elicit the peculiar sickening feeling if it is the ovary, but in the child no such sensation, if present, helps the diagnosis.

Such a swelling may be presented by a variety of conditions.

1. A superficial lymphatic gland.
2. The ovary in the sac.
3. Omentum in the sac.
4. An encysted hydrocele of the canal of Nuck.
5. A tuberculous nodule in the sac.

The ovary when present is usually mobile, and can be displaced up into the canal, while a gland has not, as a rule, so much mobility in the direction of the inguinal canal.

An encysted hydrocele of the canal of Nuck is usually only slightly mobile, and is not a common condition.

A tuberculous nodule simulates very frequently the ovary. It may be distinctly mobile, is generally situated at the apex of the sac. It is always well to examine the general abdominal condition in all such cases, although a diffuse peritoneal tuberculosis may be present, without affording any evidence on examination of the abdomen.

Hernia of the omentum is, as a rule, irreducible, and may in some cases resemble in form that of the ovary. It is not common

in children, occurring much less frequently than in the adult female.

In conclusion, the points which have struck me in considering these cases are :—

1. That inguinal hernia in the female child occurs in about 10 per cent. of all cases.

2. That it is associated in nearly 30 per cent. of cases with a descent of the ovary and Fallopian tube.

3. That the presence of these organs is often associated with malformation and defects of the genital system.

4. That the condition most likely to be confused with hernia containing the ovary is the presence of a tuberculous nodule in the sac.

References.

1. *Annals of Surgery*, 1904. 2. *Berliner Klinische Wochenscrift*, March 1906.

Meeting VIII.—May 16th, 1906

Dr J. O Affleck, *President, in the Chair*

This was a special meeting held in the Hall of the Royal College of Physicians.

Demonstration

Walter Goodenough Chase, M.D., Boston, gave a demonstration on the Use of the Biograph in Medicine, of which the following is an Abstract :—

Photography and its allied branches play such an important part in medical science to-day that we can hardly realise their value without comparing the representations in modern medical literature with those of some twenty years ago, when all illustrations had to be drawn or painted and reproduced on wood by the engraver, a most costly process. Instantaneous photography has also yielded most valuable results. The first exponent of this was Muybridge, who some twenty-five years ago startled the scientific world with his pictures of animal locomotion, for the production of which he arranged a

series of cameras, the plates in which were so timed that a series of instantaneous pictures was rapidly taken one after the other. These printed on a strip of paper and placed in the zoöpraxiscope gave the effect of motion. These proved a shock to those whose conception of animals in motion had been derived from the classic representations, the prancing steed of art being rudely transformed into an animal whose legs seemed tied into knots. Then came the invention of the moving picture, or biograph, by means of which a series of rapidly taken pictures can be so projected on the screen that the figures seem actually moving.

Some months ago it occurred to me that the biograph might be employed to depict pathologic motion, and that a series of films covering the various phases would not only be useful to the student from a diagnostic standpoint, but of value in studying and analysing the muscular action. The first step necessary in carrying out this idea was to learn the process of taking and projecting. The American Mutoscope and Biograph Company kindly put their facilities at my disposal, and instructed me in the use of the apparatus. Dr Spratling of the Craig Colony for epileptics had some time previously written them asking as to the cost of biographing epileptic subjects, but the expense had been prohibitive. The idea of reproducing various gaits had also been tried some years ago by Dr Alex. M'Lane Hamilton with the aid of a Lumière camera, and Dr Doyen's experiments in biographing surgical methods are well known. An attempt, I understand, had been made in the Salpetrière Hospital of Paris to biograph the convulsions of epilepsy. Baron Henri de Rothschild, a former student of medicine, and whose tastes ran in rather eccentric lines, had also used the biograph in showing surgical methods in the Rothschild Hospital; and at the reception following his marriage to Mademoiselle Weisweiller a biograph displayed for the guests Dr Chalot's method of curing hunchbacks.

In a recent number of a French physiological journal Anasthasius describes his method of taking a series of biographic pictures at stated intervals of a bud unfolding, and then, by a rapid projection on the screen, giving the effect of a flower suddenly blooming. He also biographed the motion of a fly, and by slowly reproducing this gave the appearance of a bird flying. Some experiments, I understand, have been

made in Germany in the reproduction, biographically, of gaits. H. M. Marvin, in 1899, carried out this idea in depicting the growth of a tree.

The most difficult pathologic movement to reproduce is that of an epileptic seizure, for the reason that it takes several minutes to set up and focus a biographic camera, and your patient is not always sufficiently obliging as to have his seizure out of doors in an available place and also at a time when the sun is at its best, for an outdoor light or a room with powerful electric lights especially arranged is absolutely necessary to good results. The only place successfully to do such work is where there is an abundance of material, as in an epileptic colony. Dr Spratling, of the Craig Colony, kindly placed the resources of the colony at my disposal, and, with the assistance of the staff, Drs Collier, Shanahan, and N. B. Ross, I have been able to successfully biograph twenty-one separate seizures, including one case of status epilepticus, which is described below.

The method may be interesting. Realising that it was impossible to set up the camera after the seizure had commenced, we had some one hundred and twenty-five male patients from the infirmary assembled in a convenient spot out of doors on a warm summer day. The clothes were removed and the patients covered with blankets, so that, a seizure occurring, the blanket could be readily dropped and the subject, within a very few seconds, placed in the range of the camera. For a background I had a large, dark brown screen made, and later a black one. The points where the head and feet of the patient were to be placed were carefully marked, and by this means the patient placed in focus, a microscopic focus on a normal subject having been previously made. It is obvious that, when the patient returned to semi-consciousness and the automatic stage commenced, the focus might vary, but later, by the use of a travelling head to the tripod, I could follow the motions somewhat.

On one side of the camera is a gauge which indicates the number of pictures per second which are being taken, and in order to have the reproduction run smoothly it is absolutely essential to turn the crank evenly. The film comes in lengths of 200 feet long by $1\frac{3}{8}$ inches in width, while the negative itself is 1 inch in width by $\frac{3}{4}$ in depth. The length of

exposure can be varied by the adjustment of the shutter, $\frac{1}{60}$ of a second being the usual length of exposure. On the film there are sixteen separate pictures to the foot, and the rate of running is from fourteen to sixteen per second.

For the reproduction on the screen, a positive is necessary, and this is made by contact printing. A sensitised film is placed over the negative and the two run automatically before an electric light. The positive film is then run through an Edison projecting apparatus which is set in front of the condenser of a stereopticon at just such a distance before the crossing of the rays that the whole field is illuminated. By the use of a water cell in the condenser, all danger of fire is avoided, as a stop of a few seconds before a plain condenser will result in an explosion. Professor Gage of Columbia has written a monograph on projection of microscopic slides, which gives most valuable points.

What is the practical application of this biographic work? First, and most important, is the educational use, both for physicians and students. The teacher, when lecturing to a large audience, can have the biographic projection, as large or larger than life size, thrown on the screen, and reproduce all the movement of rare cases, which might not occur for months in his clinic. As an instance, I have biographed one rare case of double nystagmus in which the movement of the eyes is clearly shown. Again, with the assistance of Dr Fernald of the Massachusetts Home for Feeble-Minded, I have successfully taken a group of rhythmic idiots, each with his individual motion and keeping time to music. The reproduction and analysis of the different gaits offers a wide range for the use of this instrument. On the 2500 feet which have already been taken this summer, 1500 of which were used on epileptic convulsions, there are 37,000 separate pictures, any one of which may be enlarged and the most characteristic ones mounted in series, thereby showing students the phases of the disease, where biographic projections are not feasible. I have selected a few of these to illustrate. These have been enlarged and photographed down again to blocks for printing. These enlargements may also be useful in the illustration of books.

A most novel application is the pocket biograph, one of which, representing a seizure, has just been made for me by the Biograph Company. Some sixty small prints in series are

made and bound together in small book form. Holding these by the lower side and pressing the thumb along the top, the actual motion is reproduced. Imagine the value to students or doctor while sitting quietly at home able to run through a series of these. Then again, the biogen or instrument used in the one-cent vaudeville parlours, now common in most cities, is especially adapted for showing these rare cases to students. From the ordinary biograph negative prints can be made for both these instruments, and different rolls easily substituted, as in an Æolian piano.

The case of status alluded to is illustrated by Figs. 10, 11, and 12, in both the stuporous and convulsive stages, and to my knowledge they are the only instantaneous pictures taken of such a condition. Two hundred feet of film were used in this case, occupying some four minutes in running. When it is considered that there are sixteen pictures to every foot of film, each one of which is slightly different, it will be seen that there is a total of 3200 pictures of status, an invaluable record of this condition for the student. When magnified by the biograph projection to life size, the very action of the muscles may be studied. The history of this case will prove of much interest, therefore I transcribe it, together with a slight account of the present seizure.

Patient, F. K., male, admitted to the Craig Colony, Dec. 4, 1902, at age of seven, now ten; age at onset of attacks, two. Abuse by parents was supposed to have brought on attacks. The hereditary influence was bad, parents having been alcoholic and very nervous. Attacks occurred seldom at first, and were of frequent occurrence daily at time of admission. The nature of aura unknown. Examination at time of admission showed: weight, 51½ pounds; height, 3 feet 10½ inches; hearing, sight, lungs, heart, tongue, eyes, skin, normal; speech, lisping; reflexes diminished; disorders of motility, none; stigmata; high, narrow, asymmetrical, arched palate; high forehead; broad nose; torus.

From admission, Dec. 4, 1902, to Nov. 11, 1904, he had one hundred and four seizures, commencing with seven in the first month, and increasing to twenty-seven in October 1904. In January and February 1905 he had none, and his record of seizures since is: March, 1; April, 3; May, 3; June, 3;

July, 3 ; August, 4 ; September, 2, until September 21, when this attack of status occurred.

Present attack : From 1 P.M. to 2.25 P.M. he had fifteen severe attacks, culminating at this time in status, which lasted from 2.25 to 3.20, at which time he went to sleep and slept till 5.30 P.M. On the next day patient was up and dressed, and partook of light nourishment.

Treatment : During the attack he was given three one-ounce doses of the following per rectum at 1.25, 2, and 3 o'clock P.M. : Chloral Hydrate, gr. 75 ; Potass. Bromide, gr. 180 ; Aq. Dist. ad., ℥iii.

The temperature, pulse, and respiration chart are of interest.

	Hour.	Temp.	Pulse.	Resp.	
P.M.	1.10	99.4	120	24	Sept. 21
	2.30	101.2	126	30	„ „
	3.20	101.0	132	36	„ „
	5	101.0	126	30	„ „
	8	99.4	96	24	„ „
	12	98.8	102	24	„ „
A.M.	4	98.4	96	24	„ 22
	8	98.6	90	24	„ „

Meeting IX.—June 6th, 1906

Dr J. O. Affleck, *President*, *in the Chair*

I. Exhibition of Patients

1. *Dr W. T. Ritchie*, on behalf of *Dr Gibson*, exhibited a patient, aged 58, occupied in shale-mining, suffering from SYMMETRICAL LESIONS OF BOTH THUMBS. The examination of the patient showed that there was no dissociation of sensory phenomena, sensibility to tactile, painful, thermal, and electric stimuli being everywhere normal. The reflexes and responses, as well as the initiation and co-ordination of movements, were perfectly healthy, and no other trophic phenomena were present. The case seemed to be of central but obscure origin.

2. *Mr David Wallace* exhibited a patient after CHOLECYSTECTOMY FOR SEPTIC CHOLANGITIS. Mrs J., aged 39 years, was seen by him on 13th May, and gave the following history. One week previously she had slight pain in the right side, and three days later the pain became much more severe and was accompanied by sickness and a rigor. The pain now passed downwards anteriorly to the groin. Temperature 100°. Next day the bowels moved after the administration of calomel, but the pain and sickness continued. There was no jaundice. On the following day the pain was very severe; she had three rigors and vomited frequently. The symptoms were the same on the following day, but the temperature was 101° F.

On the seventh day from the onset of the illness Mr Wallace was asked to see her as the symptoms were more severe—pain greater and now more generally over the abdomen, although the most acute pain was in the right hypochondrium. Temperature 101°, pulse 120. Sickness continued, and there was marked resistance over the right hypochondrium, but no tumour was palpable. Perihepatitis was indicated by friction.

The diagnosis of septic cholangitis was made and immediate operation advised.

Operation.—Incision through right rectus. No fluid in the abdomen, but the gall bladder found distended and dark purple. It was very tense. When opened the fluid contained flakes and had an odour characteristic of coli bacillus. There were many gall-stones in the gall bladder and one or two in the cystic duct. These were removed. The gall bladder was separated from the under surface of the liver and after ligature of the cystic duct removed. A glass tube was put down to the stump, with a loose packing of iodoform worsted around it. The wound was partially closed.

The patient made an uneventful recovery. The tube was removed on the second day and the worsted on the fourth. No bile escaped. She left hospital on the twenty-fourth day. The case is interesting from the view of diagnosis, and also as this complication of gall-stones is always dangerous.

Mr Wallace showed the *specimen* from the case. The fluid microscopically contained coli bacilli in pure culture. There were about thirty stones, but some had been broken. The mucous membrane was completely gangrenous.

3. *Mr H. J. Stiles* exhibited two children after operation for HIRSCHSPRUNG'S DISEASE.

The condition, which was also known as congenital dilatation of the colon, was named after its describer. The symptoms were obstinate constipation from birth associated with enormous distension of the abdomen. The superficial veins on the abdomen were dilated, the lower aperture of the thorax was greatly widened, and from time to time waves of peristaltic movement could be seen passing over the belly. The first case had come under observation four years ago, when the child was æt. 5, and was in all respects typical. Never since he was born had the boy had a natural motion, enemata having always been required in order to keep the bowels open. The child was in extremely poor condition—emaciated, and with deep hollows under the eyes. On opening the abdomen in the left iliac region the colon presented; it had exactly the appearance of an adult stomach. As the state of the patient precluded any extensive operation, colostomy was performed, the mucous membrane of the gut being sutured to the skin so as to make a permanent artificial anus. Two years later the child was brought back to hospital for the purpose of having the artificial anus closed if possible. On opening the abdomen in the middle line the colon was found dilated to the size of a man's thigh. As nothing radical could be done, the wound was closed. The child was now in good health and well nourished; his bowels moved daily through the artificial anus, but the intestine between the artificial anus and the rectum was found to be loaded with impacted fæces. On the whole, the result was good, because these cases generally proved fatal in early life. In the second case, operated on about three weeks ago, the typical symptoms and signs had also been present. The operation had consisted in anastomosing the ileum to the pelvic colon, so that fluid, instead of solid fæces, might enter the rectum. The disproportion between the size of the colon and the small intestine was so great as to make anastomosis difficult, and although prior to the operation every effort to wash out the colon had been made, it was found to be loaded with fluid and semi-solid fæces. After operation some anxiety was felt lest the distended bowel should press on the lumen

of the anastomosed ileum and cause obstruction, and, in fact, there were signs of this for a few days. The patient had made a good recovery and was now running about quite well. In this case the hypertrophy and dilatation was limited to the descending and pelvic colon, and this was the condition frequently met with. It was a matter of dispute whether Hirschsprung's disease was a primary hypertrophy and dilatation, whether it was due to stricture, or whether it arose in consequence of some functional derangement of the neuro-muscular mechanism of the colon comparable to what has been supposed to be the case in congenital hypertrophy of the pylorus. Probably some cases of volvulus in adults, preceded by years of constipation, were of the same nature as the condition described.

II. Exhibition of Specimens

1. *Dr G. A. Gibson* and *Dr W. T. Ritchie* exhibited a large series of tracings from four cases of HEART-BLOCK which had recently been under their observation.

2. *Mr F. M. Caird* exhibited preparations of STRICTURES OF THE COLON AND RECTUM, which had been successfully removed.

(1) Mr E., inguinal colostomy for chronic obstruction, followed in eight days by removal of a string-like carcinomatous stricture of the rectum which lay close to the floor of the pouch of Douglas. Access by mesial laparotomy in Trendelenburg position ; enterectomy ; end-to-end suture.

(2) Mr J. W., æt. 65. Inguinal colostomy for acute obstruction. Thirteen days thereafter removal of carcinoma above pouch of Douglas by mesial laparotomy in Trendelenburg position. End-to-end suture. Pouch of Douglas packed with gauze and drained. Eight days after the enterectomy a mass of fæces was found in the rectum, and on washing out the enema escaped by the open wound in the pouch of Douglas, and became freely distributed throughout the abdomen. The entire length of the mesial wound was then opened and the peritoneum cleaned and douched. It was found that the anterior aspect of the enterectomy suturing had given way. At present the patient has a suprapubic fæcal fistula.

(3) Mr J. W., æt. 57. Rectal carcinoma close to floor of pouch of Douglas. Mesial laparotomy, Trendelenburg position. Enterectomy. After removal of the tumour it was found impossible to unite the divided ends. The lower end was therefore closed and the peritoneum stitched over it. The upper end was carried out through the groin to form an artificial anus.

(4) Mrs F., æt. 54. Obstruction. Provisional colostomy in middle line in transverse colon. There was now found a limited carcinoma lying towards the splenic flexure of the colon, which was removed and the integrity of the gut restored by end-to-end suture.

(5) Mr F. S., æt. 39. Chronic obstruction. Tumour just beyond cæcum in ascending colon. Removal of parts affected, including ileo-cæcal region and 1½ foot of ileum. End-to-end suture.

(6) Mr T. M., æt. 23. Suffered from attacks simulating acute appendicitis. Extensive tuberculous invasion and stricture of ileo-cæcal region. Removal of cæcum and 1½ foot of ileum. Lateral anastomosis.

III. Demonstration

THE ANTIDOTE AND EMERGENCY CASE: A NECESSITY

By W. G. Aitchison Robertson, M.D., D.Sc., F.R.C.P., F.R.S.E., Lecturer on Medical Jurisprudence, Surgeons' Hall, Edinburgh

Amongst the impedimenta of every medical practitioner the midwifery bag maintains a paramount place. One would think little of a man who kept this requisite in bad order, or who, when the call came, had to run hither and thither collecting forceps, chloroform, needles, sutures, soap, etc. Every practical man keeps all these articles clean and in a bag or metal case ready for immediate use.

Yet, in truth, there is usually but little need for one to hurry to undertake the duties of the accoucheur. It is to be feared that in too many cases early arrival on the scene ends only in weariness to the practitioner.

On the other hand, cases of accident, suicide, or poisoning allow of no delay in their treatment. A few minutes or even

seconds may make all the difference between recovery and death of the patient. Yet I venture to say that few of us have ready at hand the means to treat such accidents. The surgeon, no doubt, has his bag of instruments ready for immediate operation, but in such cases he is rarely appealed to. It is the general practitioner who usually receives the first call to the above named cases.

The medical man may be called to a case of accident or of suicide attempted by cutting or stabbing. He would probably in such a case hurriedly collect his pocket-case, antiseptics, and dressings, and happy is his state of mind when these are sufficient. The patient may, however, have lost so much blood that recovery is very doubtful, unless aided by the injection of saline solution, and this the unhappy practitioner is unable to do, because he has not brought the appliances. Tracheotomy may be required, but again the medical attendant has not brought a tube, and the patient dies before one can be obtained.

Perhaps a message is left that some one has taken poison. Not knowing what toxic agent has been swallowed, the practitioner, after placing an antidote table in his pocket, sets out for the patient's house to discover what poison has been taken. On arrival he may be told that opium has been swallowed, and so he makes use of the ordinary domestic emetics, and valuable time is lost, as the only satisfactory antidote, namely, permanganate of potassium, has not been brought and may not be obtainable.

It is needless to multiply examples. Unless the appropriate remedies are immediately available, the patient's life may be lost. We cannot trust to chemists to supply us with remedies, as their shops are open only during the day, and a large proportion of urgency cases occur during the night,

If, on the other hand, we are told what poison has been taken before we leave our own house, then often hurried attempts are made to select certain antidotes from a cupboard too frequently overstocked with remedial agents. In the unlikely event of our finding appropriate remedies, time is wasted in weighing out the doses of each, as one would hardly like to trust to a haphazard method of administration.

To obviate all such troubles, and to ease one's mind, I would advocate the institution in every doctor's house of the

Emergency and Antidote Case. The materials for such are easily gathered together, and the drugs, with a few exceptions, keep indefinitely.

Many years ago I prepared for my own use, as well as for demonstration to my class of medical students, such a case. It was made before the employment of compressed drugs in tabloid form became general. I found that an excellent method of preserving each dose of the antidote was, after weighing it out, to cork it up in one of the small glass specimen tubes so largely employed for advertisement purposes. Each tube was labelled with the name of the substance and the dose it contained. Several doses of each antidote were prepared in this way, and the series of tubes enclosed in a small box or wide-mouthed bottle, which was itself labelled in turn. For ease in administration, it is better to group a whole class together; as, for example, one box is made to contain a series of different emetics, another various sedatives, and so on. It is wise not to rely on a single emetic or sedative, but to have several kinds of each, because the idiosyncrasy of the patient to a certain drug cannot be foretold. If, therefore, we find that a particular emetic, *e.g.* ipecacuanha, does not produce emesis after (say) two doses have been given, one ought rather to proceed with the administration of a different one, as sulphate of zinc. Harm, in the form of cardiac depression, may ensue if the emetic be not at once rejected by the stomach. In the same manner the sedative drugs exhibit a marked variation in the effects they produce on different patients. Chloral, for example, often produces great excitement unless followed by a large dose of bromide of potassium.

Having made our selection of drugs, instruments, etc., the whole may be contained in an ordinary handbag, or, preferably, in a small box, and thus all is kept ready for immediate use. On being called to a case, instead of worrying as to what remedies ought to be taken, we have only to snatch up the emergency bag, and we shall feel sure that any ordinary case will find its appropriate remedy therein. Instead of trusting merely to one's memory, it is well to include in the outfit a printed Table of Antidotes, such as are found in many of the pocket medical diaries issued gratuitously.

Only those practitioners who have fortified themselves against such sudden calls possess peace of mind akin to that

which the midwifery bag affords to the accoucheur. It is true that the case may remain unemployed for weeks together, but surely that is no argument against our being prepared for the necessity when it does arise.

Let me enumerate the contents which such a case ought to contain. Those drugs which can be obtained in tabloid form are much to be preferred. They occupy much less bulk, besides allowing accuracy of dosage without weighing. Before being administered, the tabloids should be crushed down, and, if possible, dissolved. In this way they act much more readily by neutralising the poison which has been taken. Almost all the drugs mentioned may be obtained in tabloid form.

CONTENTS OF EMERGENCY AND ANTIDOTE CASE.

1. *Emetics.*

Ipecacuanha powder given in doses of 20 grains.
Sulphate of Zinc, dose 30 grains.
Tartar Emetic, dose 3 grains.
Sulphate of Copper, dose 10 grains.
Carbonate of Ammonium, dose 30 grains.

2. *Sedatives.*

Chloral Hydrate, dose 30 grains.
Opium powder, dose ½ grain.
Potassium Bromide, dose 2 drachms.
Trional, dose 20 grains.
Chloroform.

3. *Stimulants.*

Spt. Ammon. Arom., dose ℥30 to 60 diluted.
Liquor. Ammon. Fort. (used for inhalation and in the preparation of ferric hydrate—see below).
Caffeine Citrate, dose 2 grains, either by mouth or subcutaneously.

4. *Antidotes.*

Acetic Acid (Glacial). In dilution, used for neutralising caustic alkalis.
Adrenalin solution.
Liq. Ferri Perchloridi. Used as a local hæmostatic. When diluted and mixed with Liq. Ammon. Fort.,

a precipitate of hydrated ferric oxide is formed. This precipitate when filtered off and mixed in water is an antidote in poisoning by arsenic.

Magnesia, heavy, used for neutralising acids.

Magnesium Sulphate, dose 2 drachms, used in poisoning by lead or barium.

Nitrite of Amyl capsules, dose ♏2, by inhalation; used in treatment of hæmoptysis and in aconite or chloroform poisoning.

Nitroglycerine tabloids, dose 1/100 grain.

Potassium Permanganate, dose 10 grains, or 2 grain tabloids.

Sodium Bicarbonate, dose 10 grains, used for neutralising acids.

Tannic Acid, dose 1 drachm, used in poisoning by alkaloids.

Turpentine (old or French), dose ♏30, used in phosphorus poisoning.

5. *Hypodermic Remedies.*

Apomorphin Hydroch., dose 1/10 grain.

Atropin Sulphate, 1/100 grain, used in aconite, morphin, pilocarpin, and fungi poisoning.

Cocain Hydrochl., dose 1/6 grain.

Digitalin, dose 1/100 grain, used in aconite poisoning.

Ergotinin Citrate, 1/100 grain.

Morphin Sulphate, dose 1/3 grain, used in belladonna poisoning.

Pilocarpin Nitrate, dose 1/3 grain, used in belladonna poisoning.

Strychnin Sulphate, dose 1/60 grain.

6. *Accessories.*

Saline solution, saturated and sterilised, or tabloids, for making normal saline solution for intravenous or subcutaneous injection.

Antiseptics (phenol., lysol., mercuric iodide, etc.).

Flexible collodion.

7. *Instruments.*

Bistouries, one or two.

Artery forceps, two or three pairs.

Catgut in air-tight reel.
Horsehair. Adhesive plaster in reel. Needles.
Tracheotomy tube or intubation apparatus.
Transfusion apparatus with glass or metal needles.
Syphon stomach tube with funnel.
Hypodermic syringe. Gum elastic catheter.

8. *Dressings.*
Sealed aseptic packets of dressings and bandages—one or two packets of each.
Disinfectant dusting powder (xeroform, iodoform, etc.).

An emergency case made up in the manner I have described costs but little, and is, in my opinion, invaluable.

Messrs Parke, Davis & Company have, however, lately introduced an excellent antidote and emergency case. It contains most of the antidotes which I have recommended, as well as an apparatus for saline injection, stomach tube, catheter, etc. There is ample space for instruments and dressings, and yet the case is exceedingly compact and small. At my suggestion, the manufacturers are drawing up an antidote table to indicate the method of employing the contents. I have also suggested to the firm the advisability of constructing a simple antidote and emergency case for use by the police, in cottage hospitals, and similar institutions.

IV. Original Communications

1. THE EARLY DIAGNOSIS OF GLAUCOMA BY THE SCREEN TEST—"BJERRUM'S METHOD"

By Arthur H. H. Sinclair, M.D., F.R.C.S.Ed., Assistant Ophthalmic Surgeon, Royal Infirmary, Edinburgh; Ophthalmic Surgeon, Leith Hospital.

When in the normal eye the field of vision is examined with the ordinary perimeter, the peripheral limit for white is determined by a test which subtends a visual angle of not less than $\frac{1}{2}°$. Such a test is sufficient to excite vision at the outer limit of the ordinary normal field. Some slight increase in the extent of the field—what may be called the "maximum field"—is obtained by using a test subtending an angle of $2°$, and it is usual to see a test of this kind employed for the examination

of the field in ordinary clinical work. The test commonly used with the perimeter is quite suitable for the demarcation of the absolute limits of the field in health, or for indicating areas of total loss of sight—so-called absolute scotomata—and for this purpose is a valuable clinical method.

The evidence of functional deterioration of the retina which is associated with the early stage of glaucoma occurs as a "relative defect," and it is therefore necessary to obtain a more delicate scrutiny of the field of vision than that required for the demarcation of an area of total blindness—an "absolute scotoma," as such an area is termed.

The test applied by Bjerrum's method is adapted for the estimation of the acuteness of indirect vision in zones from the immediate neighbourhood of the fixation point to the periphery of the field.

When the field of vision is examined in zones with the fixation point as the centre, it is found that the visual acuity is highest in the innermost zone, and gradually wanes towards the periphery, so that it is lowest in the outer zone. Thus, when the test-object is reduced in size so as to subtend a visual angle of less than $\frac{1}{2}^{\circ}$, the extent of the field becomes reduced accordingly. With a test subtending a visual angle of $10'$ the field is reduced to about 40°, and with a test subtending an angle of under $2'$ the field does not extend beyond about 26° from the fixation point.

When the field is examined by Bjerrum's method, the absolute limits are determined with the perimeter in the ordinary way. The screen test is then carried out for the examination of the state of relative vision over the more central zones of the field, with very small white test-objects used against a large black screen. Defects found by this test are termed relative, because of the small size of test-objects necessary for their demonstration.

The delicacy of this test results from several factors. The dull black quality of the surface of the screen forms a restful background for the patient's gaze, and a sharp contrast with the small white test-object.

The distance at which the patient is placed from the screen has a twofold effect.

Firstly, that of increasing the size to which scotomata are projected. This is a point of great importance to the clinical

worker, as it enables him to demonstrate small areas of defective vision which would otherwise escape notice, and facilitates accuracy in marking out the extent, shape, and position of such areas.

Secondly, that of reducing the visual angle of the test-object. The small size of the visual angle subtended by the test-object is the essential in this method upon which its value depends.

The apparatus.—The screen is made of black velvet—the blackest obtainable—is over 2 metres square, and is maintained in a vertical position when in use. It can be arranged to roll up like a window-blind when not required, or may be permanently stretched on a rigid wooden frame and provided with a dust sheet. Bejerrum employs two screens, either of which can be used as required. One is marked out at intervals to indicate the tangents for a test, in which the patient is placed at a distance of 1 metre from the centre of the screen. The other has the tangents marked for an examination at 2 metres. This convenient arrangement enables the tracings—which are indicated on the screen with black pins—to be directly transferred to the perimeter chart.

In all my observations I have used one screen, and have transferred the tracings to charts by reference to prepared logarithmic tables of tangents, for 1 and for 2 metres respectively. Having, during the past seven years, used the screen test in the examination of a large number of cases, I have been impressed by the advantage of maintaining the simple black surface of the velvet free from all markings beyond those absolutely necessary.

To facilitate the transference of tracings, and to enable one screen, free from all unnecessary markings, to be used for examinations at 1 or at 2 metres, I have had a measuring-rod made, which on one side shows the tangents indicating the degrees of separation at 1 metre, and on the other side at 2 metres. With the aid of this rule, the only markings necessary on the surface of the screen are the fixation point at the centre and the position of the radii, indicated by velvet buttons placed in a circle of 1 metre's radius at intervals of 15° round the margin of the screen. The screens recently provided for use in the Royal Infirmary of Edinburgh have the surface markings arranged in this way. A chin rest is employed in order to prevent movements of the patient's head.

The test-objects are discs of white ivory, and vary in diameter from 1 mm. to 20 mm. When in use, a test-object is placed in

the clip at the extremity of a long slender black rod, the "carrier," and in this way moved over the screen as desired.

The normal eye. — As has been already mentioned, a test-object subtending a visual angle $\frac{1}{2}^{\circ}$ is large enough to give the average normal limits of the field.

At a distance of 300 mm. as with M'Hardy's perimeter, a test-object of about 2.7 mm. would be of sufficient size. When the distance is increased to 2 metres, the test-object requires to be enlarged to practically 18 mm. in order to have the necessary visual angle of $\frac{1}{2}^{\circ}$.

The following are the figures which indicate the average normal field for some of the small test-objects used with the screen.

The test used in each case is stated as a fraction, the numerator of which is the diameter of the test-object in millimetres, and the denominator the distance at which the test is carried out.

FIG. 1.

	Out.	In.	Down.	Up.
White, $\frac{6}{2000}$ . . .	50°	40°	40°	35°
" $\frac{3}{2000}$ . . .	37°	30°	30°	25°
" $\frac{1}{2000}$ . . .	26°	26°	25°	24°

Individual differences occur in normal persons, so that the above figures are to be taken as general averages. The figures are in almost precise agreement with those given by Meisling, and also with Bjerrum's general remarks.

I have found ordinary perimeter charts too small for this kind of work, and have had larger charts prepared. With each bundle of fifty charts a chart is supplied showing in shaded zones a diagrammatic representation of the average normal field for four different sizes of tests. This, it is hoped, may serve as an aid in clinical work, by which results may easily be compared with the normal. (See Fig. I.)

The blind spot of Mariotte is surrounded by an amblyopic zone, which may be stated as about 1°, but which varies in extent inversely to the size of the test-object used. The outer limits of this amblyopic zone may show a regularly rounded outline, or irregularly rounded projections may be found. The contour of

LEFT. RIGHT.

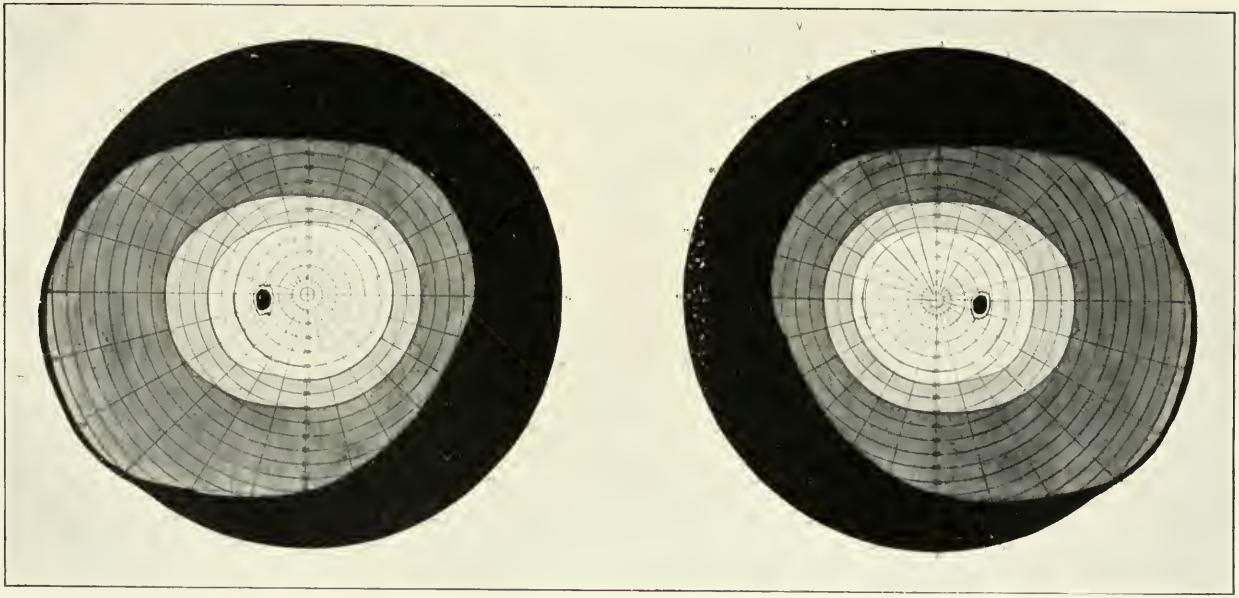

FIG. 1.—Diagrammatic Charts, right and left—referred to in the text, page 252—showing average normal field for the following tests—$\frac{10}{300}$, $\frac{6}{2000}$, $\frac{3}{2000}$, $\frac{1}{2000}$.

PLATE X.—TO ILLUSTRATE DR H. H. SINCLAIR'S ARTICLE.

LEFT. RIGHT.

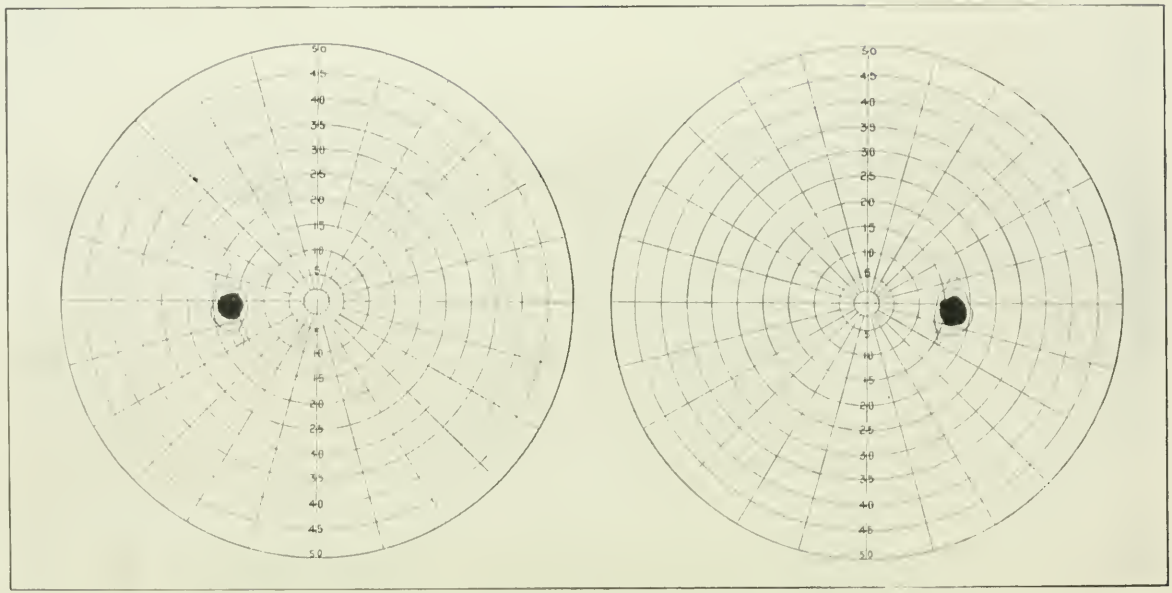

FIG. 2.—The Charts, right and left, show the normal blind spot of Mariotte as taken in the case of a young man with full vision and normal eyes. The tests used were $\frac{18}{2000}$ and $\frac{1}{2000}$. See description in the text at foot of page 252 and top of page 253.

PLATE XI.—TO ILLUSTRATE DR H. H. SINCLAIR'S ARTICLE.

the outer limits depends upon the size of the test-object and upon individual variations. The irregular extensions of the amblyopic area usually occur at the upper and the lower margins, and correspond generally to the position of the large blood-vessels as they leave the disc.

In the examples of the normal blind spot which are figured (see Fig. 2) the projections corresponding to the vessels are marked out by a dotted line, intended to indicate that the position and the limits of these projections were found to be less certain than were the margins of the amblyopic zone round the disc.

The presence of the amblyopic zone may also be demonstrated by coloured tests.

In the clinical application of this test in cases of suspected glaucoma it is necessary to have good daylight, preferably from a large window with a northerly exposure. Slight differences in illumination are not important, but when on a dull day a patient experiences unusual difficulty in locating the test-object, the examination should be postponed.

It may happen in the case of patients coming from a distance that the test has to be made on a dull day; in such case the reliability of the tracings would depend on the type of field obtained, and on the consistency of the patient's statements regarding the limits of the field. It is important to realise the difficulties which patients experience when a very small test-object is in use, and the examiner can easily satisfy himself of this by having his own field taken with $\frac{1}{2000}$.

All white or bright objects should be removed from the neighbourhood of the screen, and the walls of the room should not form too sharp a contrast with the dark screen.

The refraction should be corrected before the test is made, and this is the more necessary the smaller the test-object used.

As this test is more especially directed to the central portions of the field, the margins of the correcting glass are not likely to interfere, and in any case the glass can be tilted to a suitable position.

It is important to explain to the patient the necessity for constancy of fixation, and to keep the direction of the patient's gaze under frequent observation. In addition, replies should occasionally be verified by placing the test-object on that part

of the screen known to correspond to the blind spot of Mariotte, when, if the patient sees the test-object, it at once becomes evident that the eye has deviated from the fixation point.

Glaucoma.—When the examination is made in connection with ordinary clinical work in a case of suspected glaucoma, there is considerable advantage in testing the limits of the blind spot to begin with. The test-object used should be the smallest $\frac{1}{2000}$, if this can be sufficiently well seen, and if found to be too small the next size larger should be tried. The position and the size of the blind spot as projected on the screen depends on the distance at which the patient is placed, and can easily be determined. This having been done, the test-object selected is carried round beyond the area of the blind spot, and when seen, is then moved radially to the blind spot, over its probable limits, and these, as they are determined, are accurately marked out with black pins. Should the limits of the blind spot be traceable all round with the smallest test-object, then the case is not one of glaucoma, and does not show Bjerrum's symptom. On the other hand, should the margins of the blind spot be traced in part, and in part only, then the case is one of glaucoma, and has Bjerrum's symptom. By beginning the examination in this way time may be saved, but what is more important, the state of vision round the blind spot can be determined before the patient has been subjected to a prolonged examination. Prolonged examination is apt to develop retinal exhaustion and consequent transitory alterations in the field.

In taking the field for a small object the general form and extent should be discovered quickly by taking the principal meridians first and afterwards passing to the intermediate parts. The test-object should, as a rule, be carried from the periphery towards the centre from the area beyond the limits of sight towards the seeing area.

When using the smallest test over an area where there is a relative scotoma of very slightly lowered functional sensitivity, such as occurs where the large vessels leave the disc, I have found that the test-object can be seen when it is moved, and is lost to sight when it is kept perfectly still.

The effect of retinal fatigue must not be forgotten, especially where the smaller test-objects are used.

This method of examination was first brought into general notice by its originator, Professor Bjerrum, of Copenhagen, in 1889. In the same year, Mr Berry employed the test clinically in Edinburgh, and in the following year his clinical assistant, Dr Charles Ramage, wrote a thesis for the degree of M.D. on this subject.

Holth, of Christiania,[1] in 1896, published a paper in which he criticised some of the statements made by Bjerrum, basing his criticism on the belief that the disappearance of the test-object was mainly the result of retinal exhaustion, or, as it is called, amaurosis from continued fixation.

Meisling, of Copenhagen, in his thesis for the degree of Doctor of Medicine, published in the form of a monograph in 1899, goes into this question. He made experiments in normal persons with a test-object of 5 mm., used at 2 metres distance. The radius of the screen was traversed from the periphery towards the centre, and the test-object was moved so as to occupy eight seconds over each radius.

The fatigue phenomenon did not appear in any of the cases tested by Meisling in the way described. There was no difficulty experienced by those examined in seeing the test-object. Meisling says that his experience is in agreement with that of Bjerrum, and that he has substantiated all the statements made by Bjerrum.

In my clinical experience of this method in cases of glaucoma, I have been in the habit of indicating areas of the field in which I found evidence of retinal fatigue which was sufficient to modify the shape or extent of the field. I find, in looking over the charts of cases of glaucoma up to the end of 1904, that, out of forty-five, there are six which have dotted areas where the test-object was rapidly lost. Out of the six with fatigue areas thus indicated three figures showed the glaucoma symptom apart from the fatigue areas. In the remaining three figures the fatigue areas rendered the presence or absence of the glaucoma symptom uncertain.

The distinctive characteristic of the field in glaucoma which is referred to as "Bjerrum's symptom," depends on the relationship of the area over which vision is most fully preserved, and the area of relative defect to the blind spot of Mariotte, and may be stated as follows:—The area of normal

[1] *Norsk. Mag. f. Laegevidensk*, Christiania, No. 11, 1896.

or most fully preserved vision, as also the area of relative defect, are in contact with the blind spot, or may be said to meet at the blind spot. The conditions in the field which indicate this symptom may also be stated as follows :—The area of relative defect can always be traced up to the blind spot, provided a sufficiently small test-object be employed, and the area of most fully preserved vision is always in contact with, but does not surround, the blind spot.

The occurrence of Bjerrum's symptom may be associated with great variety in the shape, extent, and position of the relative field. Certain definite types have been recognised as being more characteristic than others. They are as follows :—

The field for a small test-object often extends in semilunar form, or as a horn-shaped projection, round the lower or the upper margin of the blind spot to the temporal side (see Figs. 3 and 4, also Fig. 6, right eye).

In some cases a semilunar-shaped field for the small test is found, isolated on the temporal side of the blind spot, with its concavity towards the blind spot.

When scotomata are present they frequently occupy a para-central position forming an arc round the fixation point, the concavity of the arc being towards the fixation point. Such scotomata never reach beyond the blind spot towards the temporal side, but may extend to the periphery on the nasal side (see Figs. 3 and 4).

The form of the field characteristic in glaucoma appears with remarkable regularity in all forms of glaucoma. It is distinctive from the field found in optic atrophy (see Fig. 5), though it may in some instances be more difficult to make the distinction than in others.

In connection with the application of this test, I have examined a large number of examples of glaucoma which were undoubted cases of glaucoma, and were recognised by unmistakable evidences apart altogether from the screen test. In all these cases, including subacute, chronic, and secondary glaucoma, I found Bjerrum's symptom present. This is valuable evidence as to the constancy with which this symptom appears in all forms of glaucoma. It is not, however, in connection with otherwise recognisable examples of this disease that the clinical importance of the screen test is to be found, but rather in the cases where diagnosis is difficult and

LEFT. RIGHT.

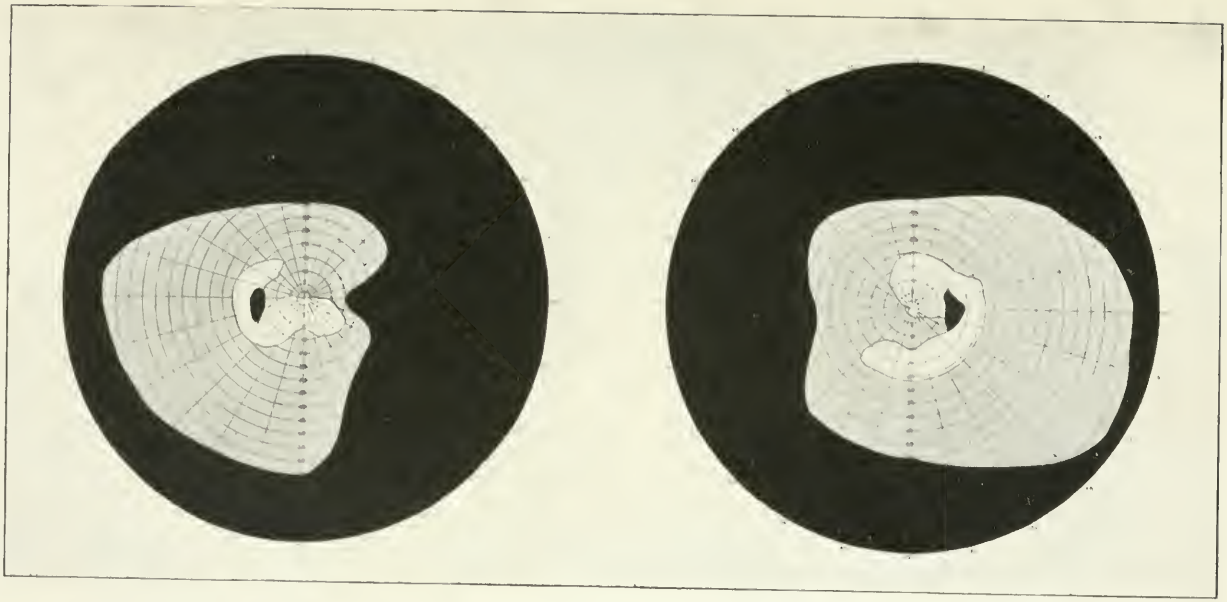

FIG. 3.—The Charts—Right V. $=\frac{6}{6}$p., and Left V. $=\frac{6}{18}$—are from a case of chronic glaucoma, and show Bjerrum's glaucoma symptom. The area of most fully preserved vision, indicated in white, is seen in contact with the blind spot of Mariotte, while the area of relative defect, shaded in grey, is also in contact with the blind spot. Note the characteristic curved or horn-shaped projection of the area of most fully-preserved vision passing round the temporal side of the blind spot. The Charts also show clearly the relative scotoma, or area of relative defect, passing in close relationship to the fixation-point, and thus giving an important indication as to treatment—see text, page 257. The tests used were Right Eye, $\frac{3}{250}$ and $\frac{1}{2000}$; Left Eye, $\frac{3}{250}$ and $\frac{3}{2000}$.

PLATE XII.—TO ILLUSTRATE DR H. H. SINCLAIR'S ARTICLE.

LEFT. RIGHT.

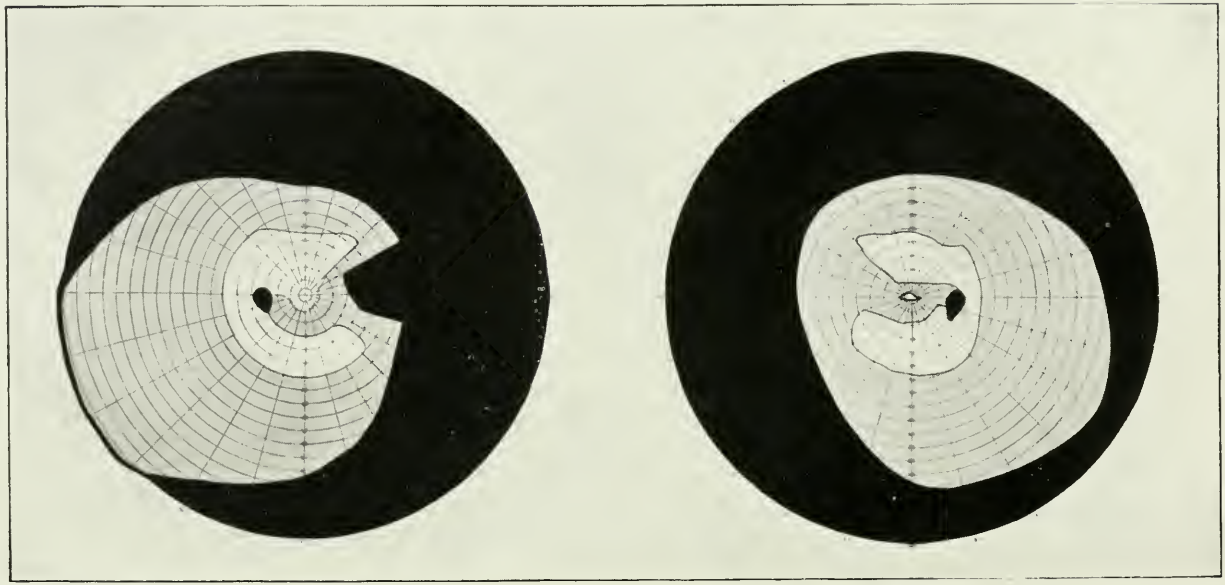

FIG. 4.—These Charts are from a case of chronic glaucoma showing typical fields. In the right eye, in which V. $=\frac{6}{36}$, a small islet of well-preserved vision remains at the fixation-point, while the relative scotoma surrounding this islet passes from the nasal side of the field to the blind-spot of Mariotte. In the left eye, in which V. $=\frac{6}{24}$, the relative scotoma passes to the blind-spot also from the nasal side of the field, forming an arc with its concavity towards the fixation-point. The tests used were Right Eye, $\frac{10}{500}$ and $\frac{2}{2000}$; Left Eye, $\frac{10}{500}$ and $\frac{2}{2000}$. The area indicated in white shows the part of the field in each eye over which a $\frac{2}{2000}$ test was seen.

PLATE XIII.—TO ILLUSTRATE DR H. H. SINCLAIR'S ARTICLE.

LEFT. RIGHT.

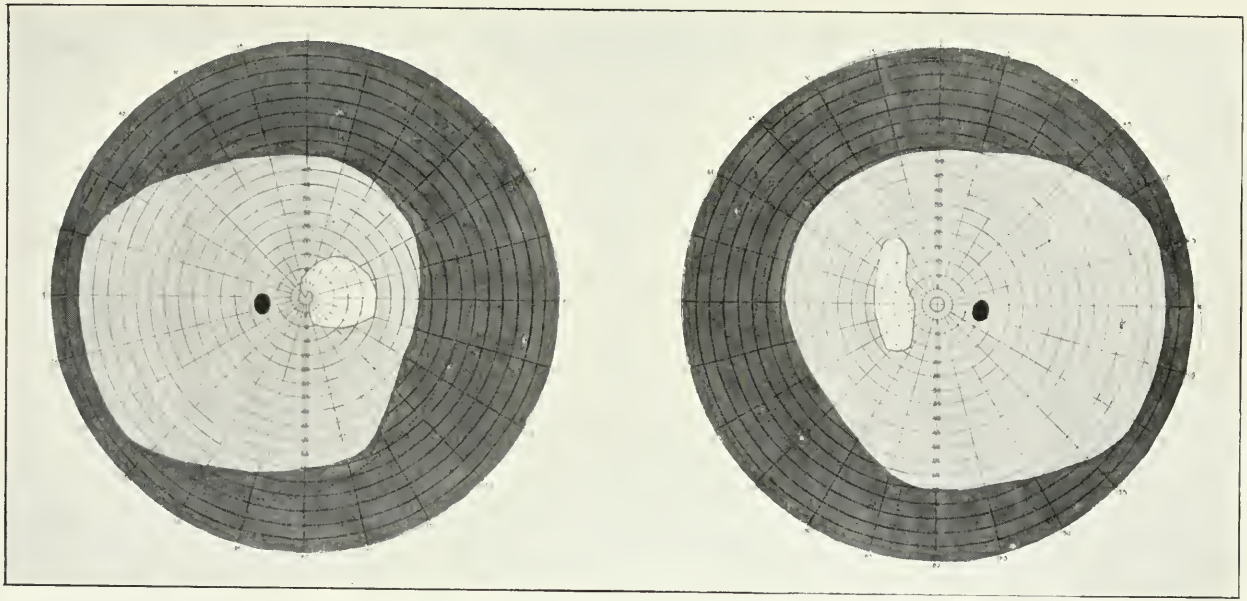

FIG. 5.—Charts from a case of double optic atrophy, showing how the area of most fully preserved vision, indicated in white, is separated from the blind spot of Mariotte, and how the area of relative defect surrounds the whole blind spot. Contrast this with one of the glaucoma charts. The tests used were in each eye $\frac{10}{300}$ and $\frac{2}{2000}$.

PLATE XIV.—TO ILLUSTRATE DR H. H. SINCLAIR'S ARTICLE.

LEFT. RIGHT.

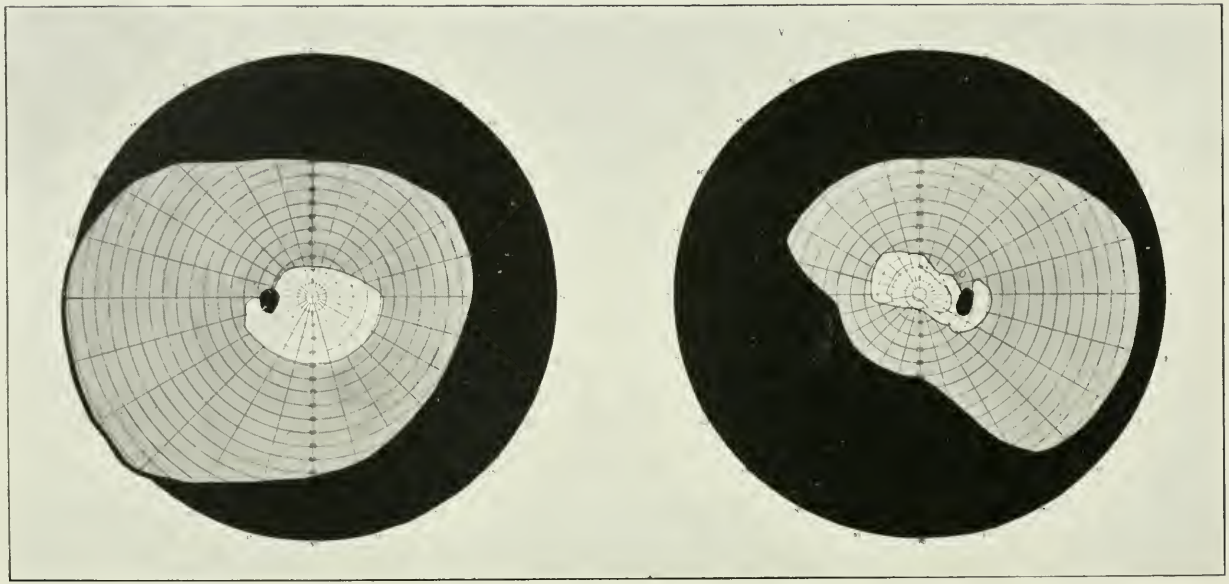

FIG. 6.—Charts from an example of chronic glaucoma occurring in a man of over seventy-five years of age. The right eye, in which V. = $\frac{6}{18}$, showed, in addition to Bjerrum's symptom, the usual evidences of well-marked glaucoma, with a history of gradual failure of central vision. The left eye, in which V. = $\frac{6}{6}$, was regarded by the patient as free from defect. Central vision was full, and the outer limits of the field were of normal extent. The only evidence of glaucoma was Bjerrum's symptom, which was quite definite, and is well seen on the Chart. The tests used were Right Eye, $\frac{5}{250}$, $\frac{6}{2000}$, and $\frac{2}{2000}$; Left Eye, $\frac{2}{250}$ and $\frac{1}{2000}$.

PLATE XV.—TO ILLUSTRATE DR H. H. SINCLAIR'S ARTICLE.

LEFT. RIGHT.

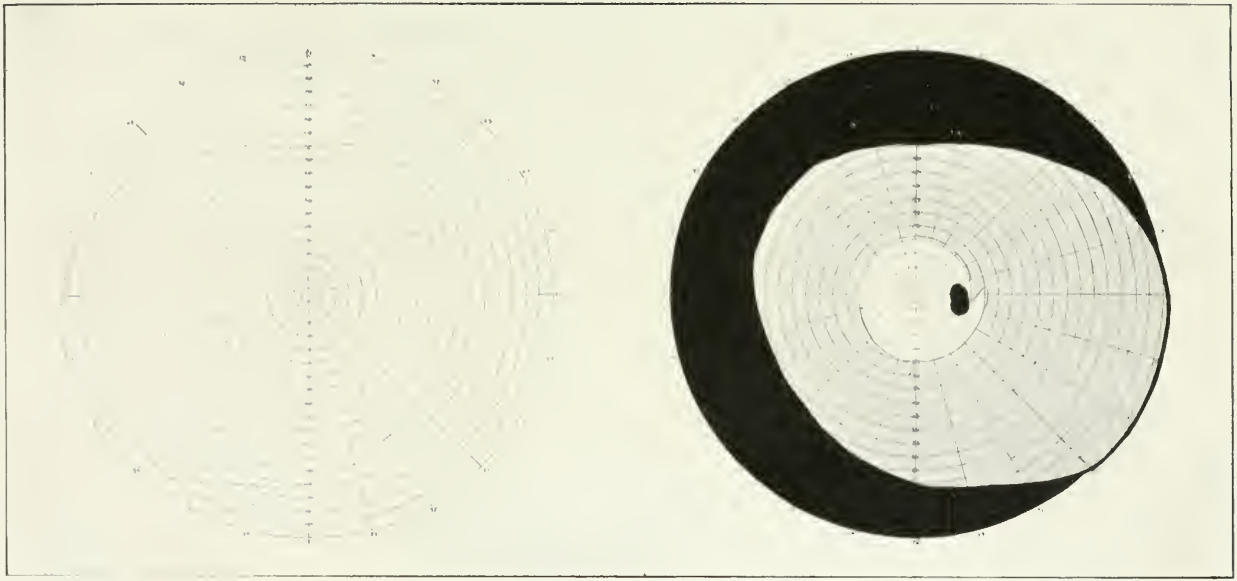

FIG. 7.—A Chart from the right eye, in which $V. = \frac{6}{6}$, and the outer limits of the field of vision were normal, showing Bjerrum's symptom as the only evidence of the presence of glaucoma. In the left eye glaucoma had resulted in entire loss of sight. The tests employed in the Right Eye were $\frac{10}{500}$ and $\frac{2}{2000}$.

PLATE XVI.—TO ILLUSTRATE DR H. H. SINCLAIR'S ARTICLE.

where demonstrable evidence is unobtainable by other means. Thus it is of special value in early diagnosis, just where difficulty of diagnosis is greatest, and where the recognition of the disease is of the first importance. The charts (Figs. 6 and 7) showing the glaucoma symptom were taken from cases in each of which one eye was manifestly glaucomatous, while the other eye showed no definite sign or symptom of glaucoma, except "Bjerrum's symptom" discovered by the screen test.

It also meets another well-known difficulty, the differential diagnosis between glaucoma simplex and optic atrophy (see Fig. 5).

In connection with treatment, too, some important evidence may be gained by the screen test. The relationship of the relative scotoma to the fixation point is a matter which in certain cases would influence one in advising the operation of iridectomy. Where the scotoma runs very close to the fixation point, or passes round it, leaving only a small islet at the centre, one would not advise iridectomy unless the state of the tension of the eye demanded it (see Figs. 3 and 4).

The time required for this test depends on the purpose for which the examination is being made. For clinical purposes, and apart from correction of refraction, twenty minutes or less is quite sufficient.

For more detailed information and a more fully illustrated account of this subject, the reader may refer to the author's paper published in the "Transactions of the Ophthalmological Society of the United Kingdom," vol. xxv.

2. VESICAL TUMOURS.

WHY OPERATIVE TREATMENT IS SO FREQUENTLY UNSUCCESSFUL IN ITS REMOTE CURATIVE RESULT. A PLEA FOR EARLY DIAGNOSIS.

By DAVID WALLACE, C.M.G., F.R.C.S.Ed., Edinburgh,
Assistant-Surgeon, Royal Infirmary, Edinburgh

BILROTH in 1874 removed a tumour from the bladder by the suprapubic route, and then performed the first operation for this disease. Albarran in 1892, and Clado in 1895 published works upon the subject which drew the attention of the profession to the importance of vesical tumours and placed the

subject upon a sound basis. They were each able to collate a large number of instances of the disease, and emphasised the fact that this was due to the increased facility of diagnosis by the introduction of cystoscopy. In the last fifteen years upwards of 700 cases of operation upon tumours of the bladder have been reported, and individual surgeons have reported as many as 100 cases. Indeed, Nitze has observed as many as 399 cases. No doubt, he has had very special opportunities, but the fact that so many have been seen by one surgeon implies that the affection is very far from uncommon. I have myself seen upwards of 50 cases.

Various classifications have been given, but that of Albarran is thought by many to be the most rational. It is unnecessary to give it in detail, but he recognises in particular papillomata and carcinomata, adenomata and sarcomata, and some rarer forms which have only occasionally been met with, *e.g.*, myomata and angiomata. Hitherto surgeons have generally believed malign tumours to be more common than benign growths, and statistics seem to corroborate this view. Thus Dr Watson of Boston, in 653 cases which he has collected, in which operation has been resorted to, found that 243 were benign while 410 were malign.

Of the benign, 203 were papillomata.

Of the malign, 358 were carcinomata and 52 sarcomata. Rafin in cases communicated to him by individual surgeons reports: *benign*, 133 cases; *malign*, 146 cases.

Nitze's cases, reported by Weinrich, give *benign* 94; *malign* 177 and 128 not exactly determined.

With the knowledge of the greater proportion of malign tumours as compared with the benign, naturally we would surmise that operative interference in many instances would be followed by recurrence of the disease, but the statistics of operation as collected by Dr Watson (*Annals of Surgery*, Dec. 1905) are much worse than the most pessimistic could anticipate. The operative mortality in *benign* cases was 17 per cent., and in *malign* 27 per cent. Then in the benign cases which survived, there was rapid recurrence in 20.5 per cent., and in the malign in 26.8 per cent.

In the statistics collected by Rafin for the French Urological Association, the results in 146 unpublished cases of carcinomata reported by surgeons were as follows: ablation of

the tumour in 57 cases gave the mortality of 14 per cent., and in the 49 survivors recurrence or death occurred in 28 during the first year. Palliative treatment was adopted in 89 patients, and few of these lived longer than six months.

In the *benign* cases, 156 suprapubic operations were performed in 133 patients, and the mortality was 6, while the later results in 91 cases gave 24 recurrences and 67 cures. Of the 67 cures, 26 were of less than one year's duration.

It must be noted here that even in benign tumours, operative interference is very frequently unsuccessful. This is probably in great part owing to the site of the tumour which, in a large percentage of cases, is in the lower zone of the bladder near the ureteral openings into the bladder. It has been said that single villous papillomata in over 90 per cent. of cases are situate not only in the above zone, but close to one or other ureteral orifice. Multiple growths are irregular in their position, but they are almost invariably grouped around the region of the trigone.

It can easily be understood how in this position tumours are difficult to reach, even by suprapubic cystotomy, so as to enable the surgeon to excise them with certainty. Portions may be left behind, and recurrence is thus almost certain to take place. This may be so when no infiltration is present and the growth single. Even in a pedunculated tumour, to ascertain, in this position, where the attachment of the tumour ends, is difficult, and when several growths are present, the difficulty is much increased. Fear of injuring the ureter must also be present in the mind of the surgeon and deter him from cutting so freely as he otherwise would do.

Another factor which increases the risk of return is the duration of the tumour since its first appearance. This is not easily estimated accurately, but approximately we can judge from the time when bleeding first occurred, hæmaturia in the majority of cases being the first symptom of simple fimbriated or warty papillomatous growth. Rafin gives statistics of 174 cases; seventy-five, to judge from the symptoms, had existed one year, while eighty had been present from two to six years, and the remaining nineteen for more than six years. It may be argued that long existence would mean slow growth, but on the other hand, that does not imply restriction of the attachment. While the outgrowth of the tumour may remain

small, we may have the deeper portion extending more widely. That the tumour will be larger and the risk of multiple growths will be greater *cæteris paribus* the longer the tumour exists, is probable, and further, if it be true, as some writers believe, that simple tumours may become malign, the longer the tumour is left the greater the risk.

It has been urged that when recurrence takes place after operation upon a fimbriated papilloma, the second growth differs in character from the first, and bears a close resemblance, in its further history, to a malign tumour. This has not been my experience. Invariably I have thought delay in growth followed operation, even though all of the tumour was not removed.

We cannot change the type of tumour we have to deal with, nor can we alter the site which it occupies, but we can lessen, I believe, the duration of its existence and thereby do much perhaps to improve operative results. Early diagnosis is for this most important and, for its attainment, a recognition of the value of cystoscopy is needed. The importance of cystoscopy as a means of diagnosis and prognosis in urinary surgery is now well recognised, but I wish to mention the importance *of its early use* in cases of hæmaturia, as in those cases due to tumour, it means early recognition of the cause. To see a tumour is easy; the examination is not painful as a rule, and can be carried out in the majority of patients without general anæsthesia. It is sometimes thought that harm may be done by such an examination, but with strict attention to aseptic measures there is practically no risk. In certain cases it is better to be prepared to operate at the time of the cystoscopic examination than to delay, but that is not necessary in all cases, and it is infinitely better to recognise the presence of a tumour and operate a few days later than to overlook the disease and allow the tumour to go on growing. Apart however from diagnosis, I wish to emphasise the value of cystoscopy in prognosis—a use of cystoscopy, in my opinion, far too little recognised. It is impossible to over-estimate the importance of noting the site and attachment of the growth, whether it be single or multiple, and if possible, by what are now fairly well recognised appearances, whether it be malignant or benign. Is it a case for curative operation, or will the only operation

suitable be palliative in its nature? This is a question of the greatest importance, as, if curative operation be impossible, we have most carefully to consider at what period a palliative procedure should be adopted. I have discussed this in a paper recently published (December 1905) in the *American Journal of Urology*. The conclusions I have arrived at and stated are: (1) Operation should be delayed, in cases unfavourable for cure, until the symptoms are so severe that the patient's life is rendered very unpleasant and his health is suffering. (2) When sepsis is present and the symptoms aggravated, and the patient suffers from septic absorption, operation is indicated. (3) When hæmaturia is persistent and the patient is losing ground, operation arrests bleeding and prolongs life. In such cases suprapubic cystoscopy is the best operation. Frequency of micturition is got rid of and the patient gains strength, as his rest at night is less broken. The permanent fistula can have an apparatus so fitted that distress from leakage of urine is almost entirely obviated.

Cancroidal tumours.—These are infiltrating tumours which invade the bladder wall and tend to ulcerate. They are practically inoperable from the time of the recognition of their presence, as has been pointed out by Guyon and others. Guyon says, "The surgeon always arrives too late." This is not necessarily because the medical attendant has neglected to try to diagnose the condition early. We must recognise that the tumour may have caused the patient so little discomfort that he has not sought advice until graver symptoms supervened, and it was then too late. How often do we see a similar unfortunate condition in tumours of the alimentary tract?

In other cases, however, and the first which I shall describe is a good example, there is much delay in a real attempt at diagnosis.

The symptoms are frequently atypical of bladder tumour, and point rather to cystitis or calculus. Pain and frequency of micturition, with pus in the urine, often constitute the first symptoms. Stone is not infrequently indicated by the passage of calcareous particles with the urine. Hæmaturia is not uncommon as an early symptom, but more usually makes its appearance after the other symptoms have existed for

some time. The patients are generally of middle age, or older, often about sixty years of age. The diagnosis may be suggested on examination per rectum when the base of the bladder is felt to be hard and fixed. Cystoscopy in these may be difficult, as the patient seeks advice late in the disease, and the bladder may be much contracted.

I have had a number of such cases under my care, and in all the history and symptoms have been very similar. The following patient may be taken as a type.

Mr C., aged 43 years, first complained of frequency of micturition in 1898. At that time he had no pain and there was no blood in the urine.

1899. Patient had pain during micturition and frequency was great; every two hours night and day. No blood.

1900. *In statu quo.*

1901. The same symptoms, but more severe.

1902. Symptoms continued, but for the first time there was bleeding.

1903. Symptoms were aggravated, and in May the bleeding was continuous for 14 days. Many clots were passed, chiefly at the end of micturition. He was sent to see me on 23rd May of that year. Bimanually there was easily palpable above and to the left of the prostate a hard firm swelling. This swelling was not irregular in shape.

Cystoscopy revealed a large tumour on the left lateral aspect of the bladder, smooth on its surface and non-ulcerated.

Suprapubic cystotomy was performed and the tumour was found incorporated with the bladder wall and obviously not removable.

The bleeding entirely ceased for a time after the operation, and the patient's general health was much improved for some weeks. He died in September of the same year.

In this case, to attempt curative interference was not justifiable. It would have meant excision of half of the bladder wall, but obviously the tumour was fixed to the parts externally to the bladder, and removal of all disease was an impossibility.

On the other hand much relief was given by a palliative procedure. As I have stated, he had temporary relief from all symptoms for a time, but died in four months.

Another patient, Mr S., aged 65, passed blood in December

1901 but had no other symptoms. For four months bleeding was intermittent, and four months later, when I saw him, he had great frequency of micturition and was suffering from septic absorption. The urine contained pus and blood. Cystoscopy showed a crateriform ulcerated surface invading the left lateral wall of the bladder. Suprapubic cystostomy was performed as a palliative procedure. Much relief to the symptoms resulted, and he greatly improved for some weeks but died four months later.

Others of the patients who come under this class of tumour improved very markedly after cystostomy, but nearly all died in less than a year after operation.

Papillomata.—In this, the most common form of benign tumour of the bladder, infiltration of the bladder wall is not present, and from the pathological nature of the growth and its anatomical attachment, successful results should be anticipated from operation ; from statistical evidence, however, such success is not even usually obtained. Two varieties of papilloma are recognised : 1. The fimbriated (villous growths) which are not infrequently pedunculated ; and 2. Warty tumours which are usually sessile, but implanted upon the mucous coat, and do not infiltrate the muscular layer.

The symptoms in these varieties are practically identical, and are now well recognised by the profession. Hæmaturia is almost invariably the first symptom, and has characters which are almost pathognomonic of this disease. It is most important, however, to remember that in some cases the bleeding does not conform to the typical form and that error may in this way arise. Not infrequently I have known cases missed in which for months, and even longer, the blood was intimately mixed with the urine and was dark in colour, giving rise to the belief that the condition was renal.

In all cases, however closely the hæmaturia may agree with our conception of that due to vesical papilloma, it is necessary to examine the bladder cystoscopically to avoid error. I have known hæmaturia from a nephroma simulate that of characteristic vesical tumour hæmaturia so closely, that, no renal tumour being palpable, the diagnosis of vesical tumour was only avoided by cystoscopy. The reverse also holds, but as a rule we see hæmaturia, which simulates renal bleeding, in ulcerated carcinomata rather than in papillomata.

Prostatic bleeding may readily give rise to the idea of vesical tumour, and *vice versâ*. In one case in which vasectomy for enlarged prostate had been performed, cystoscopy showed the presence of a well-marked pedunculated papilloma in a man 65 years of age.

To illustrate the history of a more or less typical case of papilloma, I may give the history of Mr I., aged 35, who first noticed blood in the urine one year before I saw him. There was no cause for it, and he had no other symptom. The bleeding was intermittent, and the blood, bright red in colour, came chiefly at the end of micturition. Cystoscopy revealed a sessile tumour about the size of a walnut, with short villi situate on the right lateral aspect of the bladder immediately above the ureter. Through a supra-pubic wound the tumour was removed by clipping it away along with the mucous membrane of the bladder and some muscle fibres. Microscopically it was a papilloma.

The operation was performed in 1898, and from then until now he has never had a symptom to indicate recurrence.

The following case is a very typical instance:—

Mr B., æt. 43. First bleeding in 1898. It came on without any known cause and lasted two days. There was no further bleeding for two or three years, and then from time to time he had slight bleeding without any other symptom. In January 1905 there was great hæmorrhage, and since then until the end of February the urine had never been free from blood. No other symptom had supervened. He had not had retention, and the urine contained no pus.

Cystoscopy.—Two villous tumours, one on each side of the trigone. These were removed through a supra-pubic opening, and at the present date, June 1906, eighteen months after the operation, the patient is perfectly well.

Microscopically, the tumour is a characteristic villous papilloma.

Other patients with similar histories and with tumours of a similar kind have been operated upon by me. In some, whom I have been able to follow up, no recurrence has taken place, but in others, as far as I can estimate about equal in number, recurrence has followed operation. In these I have observed at the time of operation two facts: (1) The tumours have been attached to the bladder wall more widely, and the

actual base of the tumour has been less definitely delimited than in the others; (2) These tumours have been situate, almost invariably, nearer the urethra—along, I might say, the trigone between the ureteral opening and the urethra.

In two of the cases the tumours have been multiple, which, as Nitze has said, shows at the outset a tendency to reproduction.

One of these patients seems to bear out my contention that the duration of the disease may have a deleterious influence upon operation.

J. B., æt. 45, for four years before he came under my care had intermittent hæmaturia. Renal calculus was suspected, and two years previously the right kidney was cut down upon but no stone found. The bleeding continued and increased in quantity. There was some frequency of micturition, but this seemed associated with the presence of clots. Cystoscopy revealed a villous tumour growing from close to the urethral orifice and extending on to the anterior aspect of the bladder wall. It was sessile, and a smaller one was attached close to it.

Operation.—Suprapubic cystostomy. The tumours were as seen cystoscopically, except that the second was more extensive than I had supposed. Complete removal was difficult, and I was not satisfied that I succeeded. Hæmaturia recurred in nine months. Cystoscopy showed that the tumours had recurred, and seemed more extensively to implicate the bladder wall. I advised no operation then, but said that later, a palliative cystostomy should be done, and three months later this was required. The tumours were typical fimbriated papillomata.

I believe that if we saw our cases of bladder tumours earlier, operative interference would be more likely to be successful, and that much suffering would be prevented and some lives saved. I admit the difficulty in removing the disease thoroughly, because of the site so commonly occupied by these tumours, but I think that with very free opening of the bladder, so that good access is got and the attachment of the tumour is seen, and not merely felt, we should be able to remove it completely. Trendelenburg's position and Petersen's bag in the rectum should be adopted. A head lamp to throw the light into the comparatively deep wound is most advan-

tageous. In these cases it is not an immediate bad result we have to fear, but recurrence from incomplete removal, so that the remote result is bad. Bleeding at the time can be readily controlled. That success, however, also depends in great part upon the patient being treated at an early period in the life history of the tumour seems self-evident. For the diagnosis to be made early, cystoscopic examination should be resorted to at once in all cases of bleeding from the bladder, in which no satisfactory explanation of the bleeding can be otherwise arrived at.

When the importance of this is recognised, and the harmlessness of cystoscopic examination, properly conducted, is appreciated, I believe the result of operation upon at least simple tumours of the bladder will be much more satisfactory than at present, and the heroic procedure of completely excising the bladder after a double nephrostomy and permanent drainage of the kidneys through the loins, suggested by Dr Francis Watson in the December number of the *Annals of Surgery*, will be relegated to the limbo of the "have beens."

Meeting X.—July 4th, 1906

Dr J. O. Affleck, *President, in the Chair*

I. Exhibition of Specimens

1. *Mr F. M. Caird* exhibited—

(*a*) A large vesical calculus (6 oz.) and enlarged prostate (3 oz.) removed from the same patient by the suprapubic route. Drainage was maintained by means of "Bunsen's pump," which has the merit of simplicity and ease in working. The patient was kept uniformly dry, and did well.

(*b*) Preparations illustrative of ruptured gastric ulcer and its treatment.

2. *Dr Alexander Bruce* exhibited—

(*a*) Two brains showing large sarcomatous tumours of the pituitary body, without achondroplasia.

(*b*) A brain from a case of chronic senile chorea, in which there was a thickening of the membranes of the vertex, especially along the lines of the arteries.

(*c*) A BRAIN from a case that presented symptoms of slight bulbar paralysis, in which there was arteritis of both vertebral arteries, with fibrous adhesions to the upper branches of the spinal accessory nerves.

(*d*) A HEART from a case of MITRAL STENOSIS, in which a spherical vegetation about the size of a large marble had occluded the orifice to such an extent that it admitted only a cone of about a $\frac{1}{4}$-inch in diameter.

(*e*) A case of ULCERATIVE ENDOCARDITIS, with a large vegetation lying between the aortic cusp of the mitral valve and the wall of the ventricle. During life the symptoms had suggested a cerebral complication, and lumbar puncture had shown that the cerebro-spinal fluid had contained a slight amount of albumen, and a few bacilli which stained somewhat irregularly and were mostly arranged in pairs. The fluid contained no excess of lymphocytes, leucocytes, or polymorphs. Cultures obtained from the blood showed organisms similar to those found during life in the cerebro-spinal fluid, and after death in the vegetation. During life no leucocytosis was found on examination of the blood.

(*f*) PHOTOGRAPHS of a case of PARTIAL EXTERNAL OPHTHALMOPLEGIA, showing bilateral ptosis, inability to elevate both eyeballs except to a very slight extent, very restricted movement of both eyeballs to the left, considerable pareses of both internal recti, and slight involvement of the right fourth nerve. The internal musculature of the eye was unaffected. The patient was a young girl of sixteen.

II. DEMONSTRATION

Dr H. M. Church showed and explained the METHOD OF APPLICATION OF THE TONOMETER, an instrument devised by Prof. Gaërtner of Vienna for the estimation of the arterial blood pressure. Although no instrument could give the information which the educated finger tip of the experienced physician could communicate regarding his patients' pulse, still in the matter of scientifically recording blood pressure and its variations an instrument for this purpose was necessary. Many such instruments had been devised. They were useful in recording the effects of medicine, diet, baths, muscular and mental fatigue, etc. He had compared the Tonometer with Hill and Barnard's Sphygmometer and Riva Rocci's

Sphygmomanometer, and found that in using them in the same patient under similar circumstances they had given practically the same result. None of them could be said to be scientifically accurate, but they were proximately correct. The Tonometer was more easy of application than the others, and did not in any way distress the patient. As was explained, a pneumatic ring was simply slipped over the finger and no severe compression was made on an artery.

Dr Church had taken the systalic arterial pressure of nearly every patient who had consulted him during the last few months, and had derived much aid from the study of this physical sign. The records varied from 40 to 175 m.m. Hg. in his first forty observations. In children say of 14 years of age the normal record was about 80 m.m.; in adults of about 50 years of age the normal was 120 m.m. But the blood pressure varied much, being always higher after food and after exercise, and always higher at night than in the morning. Smoking increased markedly the blood pressure. A Turkish bath diminished it 15 m.m. Butcher's meat in the diet increased it, and so on. Lewis's charts were shown indicating the variations in arterial pressure in different individuals under the same circumstances, and in the same individual under different circumstances. He considered the Tonometer an acquisition to the doctor's consulting room both for diagnosis and treatment. It was useful also at schools, especially at girls' schools, where an estimation of the blood pressure might be a guide to the amount of exercise and gymnastics suitable for the girl's strength. In young girls of the same age the arterial pressure seems to vary considerably, and is in keeping with their physical energies. He considered this latter point one of considerable importance in the practical application of the instrument.

III. Original Communications

1. RUPTURED GASTRIC ULCER IN A BOY AGED TWELVE YEARS

By G. Keppie Paterson, M.B., F.R.C.P.Ed.

The patient was a boy, aged twelve years eight months. I had attended him all his life and he had not suffered from any special ailment except recurring laryngeal catarrh. His gastric

illness began with pain, indefinitely located about the epigastric region, occurring irregularly as regards the time after food; sometimes it was half an hour or two hours, or immediately after; at times there was no pain after a meal. The pain was of a burning character; he had no gastric flatulence nor vomiting. The tongue was very slightly furred. I put him on light diet and dyspeptic remedies, but these did not help him much. These symptoms continued twelve days, when about 7 P.M., after a light dinner, he suddenly felt a very severe pain in the abdomen. I saw him an hour after and found him collapsed, pale, with slow pulse and rigid abdomen; there was no loss of liver dulness and the temperature was normal. He complained much of pain in the abdomen about the umbilicus, and almost more of pain in the left shoulder, the latter aggravated with each inspiration. The abdominal pain was relieved with a fomentation and gentle friction, and all symptoms of shock had passed off within half an hour. It looked like a case of severe colic in a child, but the shock and previous pain made me doubtful, and I left instructions to be sent for later on if he became worse. I did not see him till nine next morning. He had vomited for the first time at 9 P.M., and again once through the night, but there had been frequent retching. The pain in the abdomen had become easier during the night. There were all the signs of peritonitis present, distended abdomen, temperature raised, and loss of liver dulness in front. There was a peritoneal burst, and Mr Caird operated at 1 P.M., eighteen hours after the rupture.

On opening the abdomen, general peritonitis was found to be present, with a mass of lymph over the liver and stomach; when this was separated off, a punched-out ulcer about the size of a small split pea was detected on the anterior wall, about 2 inches from the middle of the lesser curvature. Two smears of the peritoneal fluid were taken, and afterwards one was found to be negative, the other showed a culture of staphylococci. The peritoneum was washed out and the lymph swabbed off the surface of the liver and from the pouch of Douglas. A drain was inserted in the wound and another above the pubes leading to the pouch of Douglas. He was given nutrient enemata for three days, then fed for two days partly by enemata and partly by the mouth; on the sixth day the enemata were discontinued. After the operation, and during

the subsequent lung complication, his digestion was good, and he was able to take a considerable quantity of nourishment; it was only during convalescence he showed signs of gastric catarrh. On the third morning after the operation he had a dusky hue, and his temperature was sub-normal; at 10 P.M. I found a patch of dulness with tubular breathing at the right base posteriorly. His temperature two hours before this was 98.6°, and at midnight it had risen to 100°. The dulness spread rapidly till two-thirds of the lung became involved. On the seventh day after the operation he showed signs of cyanosis and I began oxygen inhalation. His cough caused him much distress, but I was never able to get any mucus for bacteriological examination, as the sputum was small in amount and he had not the strength to expectorate it. Coughing was also sometimes attended with laryngeal spasm, as at the onset of his illness he had one of his laryngitis attacks. On the eighth day the base of the left lung became consolidated and rapidly extending involved one-third of it. His condition was now very serious. His temperature, at first irregular, began to show a morning remission, falling to normal in early morning, but rising in the forenoon and afternoon, the rising causing great distress. This morning remission did much to relieve the strain on his strength. On the ninth day after the operation, during the afternoon rise, his condition became very dangerous. His respirations varied up to about sixty a minute, the pulse was racing, at one time pulseless with marked cyanosis even though he was inhaling some oxygen; the slightest cough produced suffocative attacks with extreme dilatation of the pupils just as one meets in chloroform syncope, and there were spells of unconsciousness. Dr Affleck saw him at the time, and to all appearance he was dying. Mr Caird was hopeless. His parents were informed of his expected decease. Still we persevered. The oxygen funnel was held constantly close to his chin and mouth, and the oxygen given as fast as it would pass through the wash bottle; hypodermic injections of strychnine were repeatedly given. The spasm of the larynx was averted by applying hot sponges to the larynx as required. The temperature fell towards midnight and his condition was much relieved. Next morning, owing to the character of the temperature, the right pleura was explored with a syringe to ascertain if there was any empyema, but no pus was found. The afternoon brought a

return of the distress and anxiety, but the symptoms were not so severe. On the following four days, though always suffering from the rise of temperature, he gradually improved, and on the fifteenth day after the operation his temperature remained normal all day. His convalescence was very slow, the right lung taking a month to clear up. He is now well and strong. He had different medicines during the illness, but the very free inhalation of oxygen seemed to be the main agent in his recovery.

The cause of his illness is obscure. He had never suffered from any gastric trouble, was especially fond of broth and vegetables, and he ate slowly, masticating his food well. Previous to the onset there had not been any indiscretion in diet, and he had no knowledge of swallowing any fish bone or anything likely to injure the mucous membrane. There was slight caries of one incisor tooth, all others were in a perfect condition,—a very slight source of possible oral sepsis.

Gastric ulcer in children is a rare condition, though many cases have been recorded. Several fatal cases of hæmatemesis and of intestinal hæmorrhage, due to this cause, have been reported occurring in infants between the ages of thirty hours and fifteen days. Ulcers of the stomach have been found post-mortem at all ages in children, in an acute and chronic condition, with and without adhesions to the neighbouring organs; single simple ulcers, also multiple; by themselves or accompanying general tuberculosis and typhoid. The largest number of cases is contained in a paper by Stowell in the *New York Medical Record* of 8th July 1905. Ruptured gastric ulcer is very rare, and I have prepared an abstract of all the reported cases in children under fourteen years of age that I have been able to discover. I found fourteen cases recognised post-mortem, and only one during life besides this case. They have been arranged according to age.

Grainger Bisset (1), an infant, m., died 45 hours old, after severe intestinal hæmorrhage. Post-mortem a ruptured punched-out ulcer was found on the posterior wall of stomach, over the lesser curvature, about half inch from the cardiac opening.

Cade (2), infant, two months old, suffered from diarrhœa and vomiting. Post-mortem a single perforating ulcer was

found on the posterior wall opening into a cavity bounded by the pancreas, meso-colon, and transverse colon.

Northrup (3), f., one year old, suffering for a month from vomiting and diarrhœa. Stomach covered with small follicular ulcers, and a large one that had perforated.

Colgan (4), f., two and a half years, died of convulsions with pyrexia ; ruptured ulcer on the posterior wall near the lower border of the fundus.

Porter Parkinson (5), m., two years two months, gastric symptoms, with fever, for ten days, then hæmatemesis and acute peritonitis. Ulcer with a perforation of the size of a pin's head near the centre of the posterior wall of the stomach.

Bechtold (6), 1904, f., aged five and a half years, ill five months ; a sudden illness diagnosed as peritonitis due to appendicitis. An incision over the appendix liberated pus, but appendix was not diseased. Post-mortem a perforated ulcer was found in the greater curvature.

Reichelt (7), m., eight years ; suffered from pains in the stomach. Perforation found 1 c.m. above the pylorus in the lesser curvature.

Stowell (8), f., eight years, always delicate, recent gastric trouble with vomiting. Two perforated ulcers were found on the posterior wall about two inches from the pylorus.

Buzzard (9), f., nine years ; discomfort after food for a short time, sudden perforation and death. Rupture of ulcer in the lesser curvature of the stomach, midway between pyloric and œsophageal openings.

Barker (10), m., ten years ; three or four days ill, collapsed and died in two hours. Four ulcers in stomach, two of which had perforated.

Leith (11), f., ten years ; left pneumonia, followed by left empyema. Post-mortem, ulcer of stomach had perforated the diaphragm.

Gröss (12), f., twelve years, had acute tuberculosis. Two small and one large ulcer in stomach ; this large one had perforated into the omental, evidently shortly before death.

Rufz (13), f., thirteen years ; dyspepsia for some months. Perforation of an ulcer, two inches broad, in the lesser curvature.

Barthaz and Sanné (14), f., thirteen years, had dyspeptic symptoms and vomiting. Death very rapidly after perforation from an ulcer on lesser curvature of stomach.

These were all discovered post-mortem. The following is the record of the only other case beside my own that was discovered during life.

Cheyne and Wilbe (15), m., thirteen years; had some stomach-ache one day, which passed off with a hot-water bottle. Next day, after early dinner, he had severe pain in the abdomen and became very ill in the afternoon. He was seen by Mr Watson Cheyne in the evening, who was of opinion that it was a case of perforation of an abdominal organ, probably fulminating appendicitis. The operation for appendicitis was begun, gas escaped from the abdomen, and some whitish fluid. The appendix was found to be thickened, reddened, and contained a large concretion. It was removed, but no perforation was found in it or in the cæcum. As there were no fæces in the abdominal cavity, another incision was made over the stomach, and after careful search a ruptured ulcer was with difficulty discovered high up on the anterior surface about one inch from the cardiac end. He recovered without complication.

A case is reported in the *British Medical Journal* of October 28, 1899, of a girl, aged nine years, who had been operated on for ruptured gastric ulcer; Dr A. R. Parsons informs me it is a misprint for 19. It is unfortunate, as the case is erroneously repeated in different statistics and papers.

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2. TWENTY-FIVE CONSECUTIVE CASES OF OPERATION FOR PERFORATED GASTRIC AND DUODENAL ULCERS

By F. M. Caird, F.R.C.S.Ed., Surgeon, Royal Infirmary

Simple gastric ulcer occasionally progresses without causing any inconvenience to the patient, who may be in apparently good health, until the rude awaking due to perforation indicates the serious nature of his malady. The diagnosis of perforation is generally sufficiently obvious, and there are definite signs and symptoms which lead one to a correct diagnosis. A history of indigestion can usually be obtained, either of recent date and comparatively mild, or of long duration and often intermittent. The dyspepsia is associated with pain after food and with vomiting, which may relieve the pain. The more classic evidences of gastric ulcer, hæmatemesis and melæna, are generally, however, lacking.

Perforation may occur at any period of the day, and at once gives warning by "doubling up" the patient with a sudden agonising pain referred to the umbilical region, which causes fainting and collapse. The patient has to lie down, and generally vomits. As a rule, the passage of flatus ceases, and symptoms simulating obstruction arise. Occasionally there may be action of the bowels.

The initial condition of shock varies in degree and prolongation, and there may be a few hours of quiescence, often most misleading, followed by recrudescence of the symptoms. As a rule, however, they rapidly progress.

The pain becomes generalised ; the abdomen becomes fixed, board-like, and rigid, and no longer participates in the respiratory wave. The shabby, rapidly quickening pulse indicates danger. Respiration increases in frequency, and the temperature has a tendency to rise. On palpation there is hyperæsthesia of the abdominal wall, and there is marked local tenderness evinced in the epigastric and supra-pubic regions. The anterior liver dulness is found to be diminished, and in the course of a few hours may be entirely lost. Rectal examination reveals tenderness and bulging in the pouch of Douglas. There is not any pain on micturition. The symptoms become aggravated and soon merge into those of general peritonitis.

Some idea of the extent to which history and symptoms vary, may be gathered from the concluding table of 25 cases, of which 15 were female and 10 were male.

In each of the cases, with two exceptions, there was some antecedent dyspepsia, and in nearly half was there vomiting. Hæmatemesis and melæna occurred only once or twice. Rupture took place between early morning and eleven at night, more frequently late than early, the patients at the time pursuing their ordinary avocations or going about. In 2 cases the patient was lying quiet in bed. All experienced sudden severe pain referred to the "pit of the stomach," and in 4 cases to the left shoulder. Vomiting was present in 18 cases. The liver dulness had completely or partially disappeared in nearly every instance, and is recorded only once as normal. It is replaced by a tympanitic note due to free gas within the peritoneal cavity, or occasionally to a distended and paretic colon. There was marked localised epigastric and supra-pubic tenderness; the latter, although not always distinctly evident, is a helpful sign. Blood counts in 7 cases gave a leucocytosis which varied from 7000 to 25,000. Case 18 is remarkable for the complete cessation of symptoms and their return within six hours.

The diagnosis is sometimes obscure. There is a mimicry between certain lesions, such as perforated gastric ulcer, acute appendicitis, acute pancreatitis, and the rupture of intestinal ulcers and neoplasms.

Influenzal gastric pains, gastric spasms, and colic, as is well known, may lead the surgeon to clear up a doubtful diagnosis by an exploratory laparotomy.

The question of diagnosis and operation is determined by a consideration of the salient features presented by the sudden onset of pain, the previous history of gastric ailment, the localised epigastric and supra-pubic tenderness, the associated abdominal rigidity and pain experienced in taking a deep breath, the diminution in the liver dulness, and the increasing frequency of the pulse.

The operation should be carried out at once, no matter how unpromising the future may appear. There is no other road of escape open, and the seemingly moribund patient may be roused by intravenous saline transfusion and the use of strychnine.

The abdomen may be opened under local anæsthesia with cocain and adrenalin, but the further manipulation certainly requires a general anæsthetic. Spinal anæsthesia, as Bier at present recommends for laparotomies, by injecting tropa-cocain as high as the level of the first lumbar vertebra, may yet prove very serviceable.

The most ready access is gained by a free incision in the middle line. Œdema of the extra-peritoneal fascia may be observed. A rush of escaping gas and fluid verifies the diagnosis and rapidly relieves the embarrassed respiration.

A broad retractor introduced at the upper angle of the wound serves to raise the abdominal wall and allows us to inspect and palpate the stomach with ease. The perforation may be difficult to find. It may be very minute, concealed or plugged by lymph, or be not very accessible. It may be recognised, however, if the fluid be cleared away, by the lymph in its vicinity, by the direction from which fresh fluid or gastric content wells, and frequently it is detected by the finger. If the lesser sac be distended with fluid, the ulcer is on the posterior aspect of the stomach and is reached by lacerating the gastro-hepatic or great omentum. On identification, the ulcer should be plugged with iodoform gauze and a fresh search made for other perforations. Tapping the distended stomach or colon may facilitate the investigation.

An incision should invariably be made in the mid-line above the symphysis pubis, and a couple of Keith's glass drainage tubes should be placed in the pouch of Douglas. Into the narrower of the tubes the nozzle of a large douche is introduced, and irrigation of the pelvis with warm sterilised salt solution is maintained while the ulcer receives attention. Excision of the ulcer is not to be recommended. It means loss of time and loss of blood without corresponding gain. Closure of the ulcer is effected with fine silk. A preliminary stitch or two may be used to transfix the whole thickness of the margins, and so diminish its size. The main security is afforded by a series of interrupted Lembert sutures introduced at some little distance from the margin of the ulcer, tightened up *en masse* and then tied. Over these a second row may be needful. A trace of iodoform is rubbed in to favour plastic repair. If there is much perigastritis, with friable or unyielding tissue, it may be necessary to infold a large area of

the gastric wall, and as this might occlude the gastric outlet, the operation of gastro-jejunostomy may prove essential, and should be performed forthwith. On three occasions this was found necessary. The whole region of the upper abdomen should now be carefully irrigated, and every peritoneal recess douched till the return saline comes clear. In some cases we

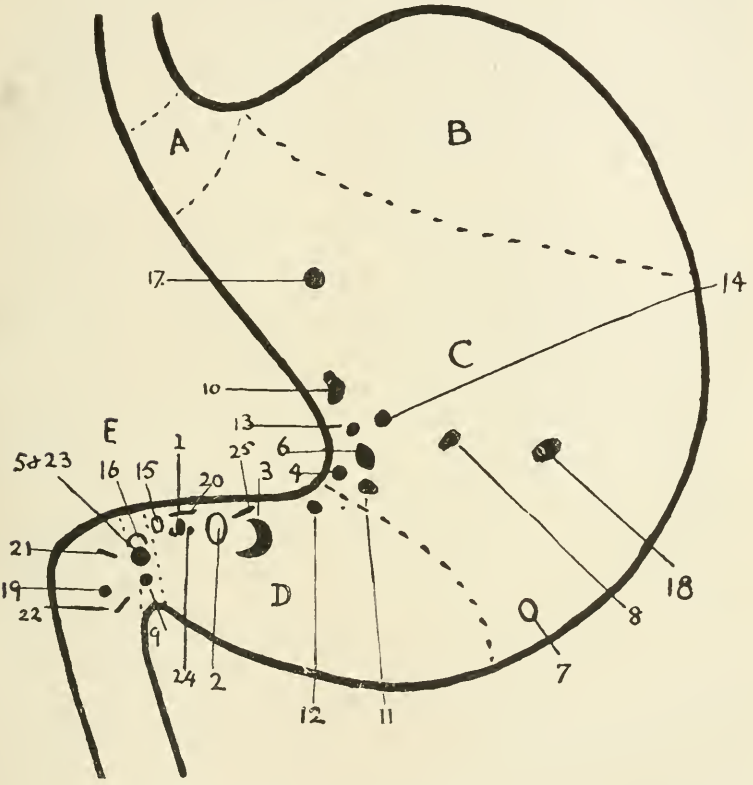

Diagram of stomach to show sites of perforation.—A, Cardiac portion; B, Fundus; C, Middle; D, Pyloric portion; E, Pylorus. Gastric ulcers posterior, 2, 7, 15, 16. Duodenal, 19, 21, 22.

have simply swabbed out the region of the ulcer with moist gauze, but have never omitted careful pelvic irrigation. A large Keith's tube is left in the pouch of Douglas, and we have, in addition, generally drained the site of perforation with a Mikulicz tampon and drainage tube. The wound is now closed. The head of the patient's bed should be raised about six inches in order to favour the gravitation of any discharge

from the dangerous absorptive region of the diaphragm towards the less susceptible pouch of Douglas.

Perforations leading to acute symptoms occur mainly in the anterior aspect of the stomach, where there is less chance of adhesion to neighbouring structures. They are most frequent towards the lesser curvature and the pylorus. Of our twenty-five cases of perforation, eighteen were anterior, four were posterior, three were duodenal (see Figure). There may be more than one perforation. The ulcer varies in type from the characteristic small, sharply cut, terraced form with comparatively healthy surroundings, to the large, ragged rent in the midst of a chronic indurated perigastritis with œdematous serosa.

It would appear that as the acrid stomach contents escape into the peritoneal cavity that the whole serous membrane reacts, and rapid effusion of an alkaline nature takes place which neutralises the acid secretion. The greater part of the intra-peritoneal fluid discovered at operations is due to this source. Blue litmus paper acquires a deeper tint on contact with it, but reddens at once on touching the fluid at the very margin of the perforation. The duodenal ulcers, which tend to affect the anterior aspect of the first part, close to the pylorus, exude a mucous with a strongly alkaline reaction.

Recovery is frequently uneventful. The chief factor affecting the issue is the nature of the organisms liberated within the peritoneal cavity and the power of tissue resistance to the toxins evolved. Fortunately we do not meet with organisms of excessive virulence within the stomach, and when there is no stagnation or fermentation of the gastric contents, the highly acid nature of the gastric secretion which we so frequently meet in such cases inhibits the growth of most microbes. An additional source of sepsis may be due to the damage inflicted on the visceral peritoneum, permitting the passage of intestinal organisms such as the colon bacillus. Hence the value of a speedy recognition of the serious nature of the lesion, and the success which is likely to crown an early operation.

Cultures were made from eleven cases, and micro-organisms were present in seven. They varied in kind. The colon bacillus was frequent. In four the result was negative. Gelatine and Agar tubes were employed, and this somewhat

crude method, together with delay in incubation, may account for the tubes remaining sterile.

In considering the prognosis, we naturally take into consideration the size and position of the perforation, the date and the nature of the last meal, and the amount of material which escapes into the peritoneum.

There can be no doubt also as to the truth of the general statement that the longer the period which has elapsed since perforation, the graver is the outlook, although it fortunately did not hold good with our cases. Apart from this, more valuable prognostics are to be gained from the age of the patient, from the rate and condition of the pulse and the respiration. A pulse of 120 is to be feared, of 130 to be dreaded. The temperature is of less moment.

The aftercourse may be one full of anxiety, requiring extreme watchfulness and care, and is conducted on the lines common to abdominal operations. The pronounced thirst due to the loss of fluid by the peritoneal effusion may be allayed by rectal salines administered every four hours. The urine should be carefully examined and morphine given as indicated. If the pulse fail in strength and fulness and increase in rate, intravenous saline transfusion is most beneficial. One litre may be given, and when improvement is maintained thereafter for a couple of hours, transfusion may be freely repeated with every prospect of success, even if there be a subsequent flagging of the pulse.

Vomiting, if severe, persistent, or accompanied by hæmatemesis, is best combated by gastric lavage.

Pulmonary complications were responsible for some fatalities during the aftercourse. One case was tapped successfully for pleurisy, and two recovered after resection of portions of rib for empyema. The last of the three cases (18) was readmitted three months later, and succumbed to an intracranial abscess which we failed to discover on trephining.

The statistics which are added, founded on such a small number of cases, must be taken for what they are worth. They seem to illustrate, however, the relation of the rates of pulse and respiration as influencing the prognosis. It will be seen that the mortality is not excessive, and that an early diagnosis may do much to render operation for gastric ulcer highly successful. It was impossible to save such cases as 16, who

already had advanced congenital cystic disease, and who sank from uræmia; and 15, whose age, 71, and previous intemperance, precluded a hopeful result. There is no reason why the judicious country practitioner should not himself act in such emergencies, and by a comparatively simple operation, not in itself dangerous, save lives that otherwise might incur still greater risk were they sent to a far distant hospital.

The concluding table may be referred to for additional information as to the individual cases.

I desire to express my thanks to the gentlemen who sent the cases, and to the pathologists of the Royal Infirmary who kindly superintended and examined the cultures.

Statistics.

I. Twenty-five consecutive cases of perforated gastric and duodenal ulcers—

Gastric, anterior	18
,, posterior	4
Duodenal	3

II. General mortality, 36 per cent.—

Males	10	4	died
Females	15	5	,,

III. Age—

12½ to 20 years	3 cases	1 died	Mortality,	33 per cent.
21 ,, 30 ,,	11 ,,	1 ,,	,,	9½ ,, ,,
31 ,, 40 ,,	9 ,,	5 ,,	,,	55 ,, ,,
41 ,, 71 ,,	2 ,,	2 ,,	,,	100 ,, ,,

IV. Time between perforation and operation—

3 to 12 hours	7 cases	3 died	Mortality,	42 per cent.
13 ,, 24 ,,	13 ,,	5 ,,	,,	38 ,, ,,
25 ,, 51 ,,	5 ,,	1 ,,	,,	20 ,, ,,

V. Pulse rate—

76 to 120	13 cases	2 died	Mortality,	15 per cent.
121 ,, 130	6 ,,	2 ,,	,,	33 ,, ,,
Above 131	6 ,,	5 ,,	,,	83 ,, ,,

VI. Time and average pulse—

3 to 12 hrs	7 cases	P. 111	3 died	P. 117	4 recovered	P. 107
13 ,, 24 ,,	13 ,,	,, 126	5 ,,	,, 141	8 ,,	,, 117
25 ,, 51 ,,	5 ,,	,, 114	1 ,,	,, 128	4 ,,	,, 111

VII. Time, average pulse, and respiration rate of 23 cases—

Time.	Cases.	Av. Pulse.	Av. Resp.	Re-covered.	Av. Pulse.	Av. Resp.	Died.	Av. Pulse.	Av. Resp.
3 to 12 hours	7	111	31	4	107	28	3	117	35
13 ,, 24 ,,	10	122	30	6	114	27	4	134	36
25 ,, 51 ,,	6	123	32	4	111	33	2	148	30

	Name, Age. Occupation. Date of Operation. Event.	Previous History.	Onset.	Provisional Treatment.	Condition Prior to Operation.	Local Condition.	Operation.	Aftercourse.
1	Mr W. L., 21; cabinet maker; 16, xii, '01; 26 hours; recovered.	Indigestion, several years, pain across stomach for 3 weeks.	Sudden, 2 hours after heartybreakfast, severe pain causing him to fall down; vomited shortly after and later twice or thrice.	Opium by mouth, and morphine.	P. 100; T. 108.8; R. 26; pain and tenderness over recti, especially tender above and to right of umbilicus; liver dulness less, quite gone within an hour; rectal tenderness.	Small ulcer, anterior near pylorus; perigastric clear fluid and lymph.	$CHCL_3$; upper part of abdomen dried out; pelvis washed out; drainage.	Good recovery.
2	Miss A. G., 23; domestic servant; 14, x, '02; 50 hours; recovered.	Two years ago, pain after food and anæmia; well since.	Sudden pain at left costal margin, left shoulder, and axilla, when lifting a couple of pails: had to lie down; vomited once blackishfluid.	Whisky; poultices.	P. 125; T. 99; R. 26; tumid abdomen; general tenderness, maximum at left costal margin.	Large ulcer near pylorus on posterior wall.	Ditto.	Good recovery.
3	Miss L. T., 22; domestic servant; 21, iv, '03; 4 hours; recovered.	Five years' indigestion; hypogastric pain for 3 days.	Sudden.		P. 126; T. 100.6; R. 30; general acute tenderness; no liver dulness.	Large ulcer on anterior wall near pylorus and lesser curvature; much clear fluid, lymph and potato.	Whole peritoneal cavity swabbed out; drainage.	Good recovery.
4	Miss M.Y., 21; housemaid; 22, ix, '03; 24 hours; recovered.	Three years' dyspepsia; hæmatemesis at NewYear; passed blood by rectum from 17th to 20th September.	Sudden, violent pain and vomiting; action of bowels this morning.	Morphine.	P. 128; T. 100.6; R. 24; great abdominal pain, felt also in chest and shoulders; rigid abdomen; general tenderness pronounced above umbilicus; liver dulness much diminished.	Small round ulcer, anterior, near lesser curvature, five inches from pylorus; slight tendency to hour-glass contraction.	Ditto.	Good recovery; symtoms again came on; fresh rupture, 3, xi, '04, see Case 11.
5	Mrs W., 37; 25, xi, '03; 17 hours; died.	Indigestion all her life; great tea-drinker; during last six months increased pain radiating from the back to the abdomen, relieved by vomiting.	Sudden pain and collapse.		P. 145; T. 103; R. 40; very tender tumid abdomen; no liver dulness.	Large ulcer, anterior aspect of pylorus.	Laparotomy with cocain and adrenalin; $CHCL_3$ in small amount; posterior gastro-jejunostomy since pylorus was narrowed after suture of ulcer; ditto.	Improved somewhat; then had to be transfused; died twelve hours after operation. Sectio: Roughening of diaphragmatic pleura.

	Name. Age. Occupation. Date of Operation. Event.	Previous History.	Onset.	Provisional Treatment.	Condition Prior to Operation.	Local Condition.	Operation.	Aftercourse.
6	Miss M'E., 25; 31, i, '04; 18 hours; died.	Old indigestion.	Sudden, severe pain		P. 124; T. 99.8; R. 36; does not look very ill; tender epigastric and supra-pubic regions; groins less so; rigid abdomen.	Small perforation, anterior, near lesser curvature.	$CHCl_3$; whole peritoneal cavity washed out; drainage.	Did well till following afternoon, when pulse rose, coffee ground vomiting came on; pelvis washed out; result negative; died 18 hours after operation.
7	Miss N. B., 25; domestic servant; 20, ii, '04; 10 hours; recovered.	For some years pain and vomiting after food; very anæmic during last 2 months; has vomited after every meal last week.	Awakened by sudden epigastric pain and bilious vomiting; bowels have acted once.		P. 102; T. 100.2; R. 24; abdomen rigid; general tenderness, marked in epigastrium; liver dulness slightly less; some rectal tenderness; leucocytes 7000.	Small ulcer, posterior, near greater curvature in lesser sac distended with fluid; no fluid in general cavity.	Ditto.	Sickness and hæmatemesis next day; stomach washed out; vomiting ceased; transfusion necessary at night; good recovery.
8	Mrs H., 40; 13, iv, '04; 6 hours; died.	Pain after food and vomiting for some time.	Sudden epigastric pain and collapse.		P. 140; T. 100.8; R. 40; abdomen rigid and exceptionally tender.	Small ulcer, anterior aspect cardiac end, midway between the curvatures.	Ditto.	Transfused at intervals of 4 to 6 hours; she held her ground for 24 hours and then sank, 32 hours after operation. Sectio: general peritonitis.
9	Mr M. Y., 48; labourer; 26, x, '04; 24 hours; died.	Eight years' indigestion; pain and vomiting after food; intemperate; a daughter died of perforated gastric ulcer.	Sudden vomiting of and passage of black matter at stool; acute epigastric pain.		P. 132; T. 97; R. 30; fixed abdomen, tender, especially over pubis; no liver dulness.	Small clean cut ulcer near posterior aspect of pylorus; coffee-ground like fluid in peritoneum; gangrenous omentum.	Distended stomach emptied with trocar and cannula; gangrenous patches removed; ditto.	Transfused after operation; broncho-pneumonia, purulent sputum, rise of temperature; death on 7th day.
10	Miss Y., 27; 18, x, '04; 17 hours; recovered.	Has been under treatment for gastric neuralgia; pain after food.	Sudden epigastric pain and vomiting.		P. 120; T. 100; liver dulness diminished.	Small ulcer near lesser curvature and pylorus.	Ditto.	Good recovery.
11	Miss M. Y., 22; housemaid; 3, xi, '04; 19 hours; recovered.	See former history, Case 4; good health since operation for perforation; quite recently pain after food and vomiting.	Severe abdominal pain and vomiting.		P. 104; T. 98.4; R. 24, at 11 A.M.; did not look ill; slight abdominal pain and tenderness; abdominal respiration; liver dulness normal; at 3.30 P.M. P. 110, T. 101, R. 28; liver dulness gone.	Ulcer near old site; marked hour-glass contraction; many omental adhesions and bands.	Ditto; posterior gastro-jejunostomy.	Good recovery.

	Name. Age. Occupation. Date of Operation. Event.	Previous History.	Onset.	Provisional Treatment.	Condition Prior to Operation.	Local Condition.	Operation.	Aftercourse.
12	Miss B. K., 21; 19, xii, '04; 48 hours; died.	Epigastric pain after meals, shooting to back and shoulders, followed by vomiting of indigested food, eighteen minutes' duration; better for a year, worse for last 12 months.	Severe epigastric pain and vomiting.		P. 168; T. 100.2; R. 32; whole abdomen very tender, tumid; no liver dulness.	Large ulcer near middle of lesser curvature; much fluid; flakes of lymph and food stuff.	$CHCl_3$; peritoneum washed out; drained.	Death.
13	Miss H., 34; 17, i, '05; 9 hours; recovered.	Two years ago had for two months pain after food; good health since; 14 days ago slight return of pain.	Sudden severe epigastric pain.		Pulse 100; T. 100; R. 28; anxious expression, rather rigid abdomen; pain in hypo-gastric and left hypochondriac regions; maximum tenderness between umbilicus and ensiform cartilage and above pubis; liver dulness gone.	Small ulcer near lesser curvature towards cardiac end: œdematous extra peritoneal tissue	Ditto.	Good recovery.
14	Master A. P., 12½; 15, ii, '05; 18 hours; recovered.	Pain after food for last 12 days, and slight cold.	Felt very ill; no great tenderness; vomited, but passed a quiet night.	Hot fomentations.	P. 130; T. 101.2; tender abdomen; no liver dulness.	Small punched out ulcer near middle of lesser curvature.	Ditto.	Signs of right basal pneumonia on 3rd day, on 8th day both lungs affected; P. 126, T. 103, R. 48; after an anxious time got well; oxygen inhalation.
15	Mr T. W., 71, dairyman, 14, iv, '05; 9 hours; died.	Three years ago an attack of vomiting, pain, and constipation; one year ago a second; has an umbilical and double inguinal hernia.	Sudden sickness and vomiting of reddish material.	Opium.	P. 112; T. 98; R. 36; tenderness above and below umbilicus; liver dulness diminished.	Posterior aspect of pylorus, large ulcer; gas, fluid, and gastric contents in quantity. Culture: colon bacillus.	Ditto.	Died 8 hours after operation. Sectio: peritonitis.
16	Mr J. F., 32, labourer, 5, v, '05; 18 hours; died.	Intemperate; no data.	Sudden abdominal pain; distension.		P. 136; T. 98; R. 40; patient had a peculiar dazed look; was not clear-headed, and had dusky complexion.	Ulcer on anterior aspect of pylorus; much fluid; pure cultures colon bacillus.	Ditto.	Rapid sinking; symptoms of uræmia; bladder empty. Sectio: advanced congenital cystic disease of both kidneys, cysts in pancreas and mesentery, hypostatic congestion both lungs, peritonitis.

	Name. Age. Occupation. Date of Operation. Event.	Previous History.	Onset.	Provisional Treatment.	Condition Prior to Operation.	Local Condition.	Operation.	Aftercourse.
17	Miss C. K., 35; 2, iv, '05; 13 hours; recovered.	For the last 14 years intermittent attacks of sharp pain after meals in epigastrium and between the shoulders; vomiting since Xmas; well till last night; drinks much tea.	Sudden epigastric pain and vomiting.		P. 120; T. 99; R. 26; abdominal tenderness; liver dulness less.	Ulcer anterior aspect; cardiac portion, 2¼ inches from lesser curvature; cultures from near rupture gave many colonies of streptococcus pyogenes, and a few of staphylococcus aureus; from the pouch of Douglas, pure culture staphylococcus pyogenes.	$CHCl_3$; peritoneum washed out; drained.	Left sided pleurisy; aspirated 10th June; Frænkel's pneumococcus; improvement; empyema followed; resection of rib; strept. pyogenes; good recovery.
18	Mrs C., 29; 21, vii, '05; 14 hours; recovered.	For several years pain after food, relieved by meal; well since March.	Sudden sharp shooting pain epigastric and umbilical regions and down left upper arm; symptoms passed off completely, but returned worse in 6 hours.		P. 140; T. 100; R. 24; pinched look; knees drawn up; tense painful abdomen; most tender in left hypochondriac and iliac regions; liver dulness normal; leucocytosis, 8000.	Large ulcer middle of anterior aspect rather nearer greater curvature; much gastric content and fluid; bacillus of colon type in culture from pouch of Douglas and from near stomach; from latter site also a few cultures of a streptococcus.	Ditto.	Left sided empyema; resection of rib; good recovery. [Readmitted October suffering from intracranial abscess; unsuccessful trephining; died.]
19	Mr R. J., 21, flydresser; 24, x, '05; 10 hours; recovered.	Good health; appendix removed June 1905.	Sudden violent abdominal pain, almost fainted; seen at once; P. 97. small, thready; T. subnormal; within an hour liver dulness gone; vomited twice.	Morphine Hypodermically.	P. 102; T. 98; R. 32: shallow thoracic; general tenderness in lower part of epigastric region; not marked in supra-pubic region; general tympanitis; no liver dulness.	Minute aperture on anterior aspect of duodenum close to pylorus; serous fluid and flakes of lymph; omenta œdematous; culture negative.	$CHCl_3$; region of ulcer swabbed; pelvis washed out and drained.	Good recovery.

	Number. Age. Occupation. Date of Operation. Event.	Previous History.	Onset.	Provisional Treatment.	Condition Prior to Operation.	Local Condition.	Operation.	Aftercourse.
20	Mr C. S., 40; 11, xi, '05; 18 hours; recovered.	Periodic attacks of indigestion and vomiting every six weeks for past 13 years; never vomited blood.	Doubled up with sudden violent epigastric pain; shortly after vomited; slept 1½ hours, arose, and pain and vomiting returned; 3 hours after attack, P. 120; full bounding rigid abdomen; vomited all night.	Hot fomentations; laudanum internally.	P. 120, weak, intermittent; T. 98.3; R. 44; abdomen rigid, fixed, slightly tumid below umbilicus; general tenderness, excessive in epigastric and supra-pubic areas; liver dulness diminished; peculiar "boxy" note all over abdomen; extremities cold and blue.	Small clean cut ulcer on anterior aspect of stomach ½ inch from pylorus; œdema of extra peritoneal tissues; much yellow sero-purulent fluid; viscera covered with lymph, a patch on upper surface of the liver. Cultures: peritoneal, yeast forms; pouch of Douglas, yeast forms, colon bacillus, diplococcus like pneumococcus.	Ditto, as above; region of wound, as well as pouch of Douglas, drained.	Pulse barely perceptible at end of operation strychnine and digitalis 3 hours after, 140; intravenous saline; pulse fell to 120; cough, expectoration, diarrhœa 3 weeks after right pleural sac aspirated on 2 occasions, yielded 2 and 10 oz. slightly turbid fluid; eventual recovery
21	Mr G. A. F., 22, hotel boots; 19, xii, '05; 26 hours; recovered.	Good health.	Sudden severe pain just above umbilicus; suffered all night; vomited 3 times black material; retention of urine; no passage of flatus.		P. 102, strong, regular; T. 100; R. 56; rapid, shallow, jerky, painful; abdomen not tumid, rigid, board-like; marked tenderness in epigastric and right hypochondriac regions; slightly tender in left lumbar, but not tender in supra-pubic region; tenderness and downward projections on rectal examination; no liver dulness; leucocytes 16,000.	Punched out oblong ulcer with friable margins on anterior aspect of duodenum close to pylorus; much gas and fluid; distended stomach punctured with trocar and cannula, 6 oz. content and this escaped; ulcer very inaccessible, covered by liver.	Cocain and adrenalin followed by $CHCl_3$; peritoneal cavity washed out; wound and pouch of Douglas drained.	Good recovery.
22	Miss G. B., 27; 25, xii, '05; 31 hours; recovered.	For years has had pain after food; past year in better health.	Doubled up with sudden pain low down on right side of abdomen; vomited all night; better next morning, till pain and vomiting returned; no passage of flatus.		P. 118; T. 99; R. 26; shallow, thoracic; abdomen slightly tumid; general tenderness, maximum above umbilicus and in right iliac fossa; some supra-pubic tenderness; liver dulness gone; marked tenderness and slight boggy feeling per rectum; leucocytes 25,000.	Ulcer, oblong, ½ inch, towards lower margin first part of duodenum; much gas and turbid fluid; that in pouch of Douglas less purulent than usual. Cultures: sarcinæ, various bacilli and diplococci like pneumococci.	$CHCl_3$; peritoneal cavity washed out; pouch of Douglas drained only.	Good recovery.

	Name. Age. Occupation. Date of Operation. Event.	Previous History.	Onset.	Provisional Treatment.	Condition Prior to Operation.	Local Condition.	Operation.	Aftercourse.
23	Miss M. S., 31, office; 16, i, '06; 10 hours; died.	Indigestion for many years; during last month on milk diet.	Seized with sudden abdominal pain when at work; vomited twice.		P. 100. rather feeble, small volume; T. 99.4; R. 30; shallow, halting, painful; abdomen rigid, hard; epigastric tenderness in above umbilicus and supra pubic; right lumbar and iliac regions tender; general tympanitis; liver dulness gone; rectum tender, no boggy feeling; leucocytes 14,000.	Round punched out ulcer, friable margins towards upper anterior margin of pylorus; much serous flake laden fluid; the narrowed pylorus so reduced by suturing, posterior gastro-jejunostomy performed. Cultures: nothing developed.	Ditto; peritoneal cavity washed out; wound and pouch of Douglas drained; posterior gastro-jejunostomy.	Did well till 4th day; then followed refusal of food, hallucinations and delirium; P. 120; T. 102; death on 10th day Sectio: no peritonitis; consolidation of upper lobe and appearance like confluent broncho-pneumonia; some acute pleurisy; left lung some congestion and œdema; liver, fatty changes; kidneys, fatty; some degree of chronic nephritis present.
24	Mr S. N., 35, upholsterer; 17, ii, '06; 24 hours; recovered.	Pain 2 or 3 hours after food for 4 or 5 years; frequent coffee ground vomit; has been treated for gastric ulcer; a heavy drinker for 5 years.	Sudden general severe abdominal pain when lying ill in bed; vomited 5 or 6 times; easier when lying on left side; flatus passed after enema.	Morphine.	P. 76, and strong; T. 98; R. 20; tumid abdomen; restricted motion; marked epigastric, umbilical, and supra-pubic tenderness; maximum just above umbilicus; tender in right lumbar and iliac regions, and slightly on left side; distinct superficial succussion and splashing sound elicited between umbilicus and pubis; face pinched, hands cold; leucocytes 8000.	Longitudinal ulcer, ¾ inch, at upper margin of stomach close to pylorus; pylorus narrowed, dense cicatricial contraction; much blackish coloured fluid; stomach emptied by puncture, and posterior gastro-jejunostomy performed.	Ditto; peritoneal cavity washed out; wound closed; pouch of Douglas drained; posterior gastro-jejunostomy.	Good recovery.
25	Mr A. M., 35, painter; 1, v, '06; 51 hours; died.	Gnawing abdominal pain off and on from Oct. '04 till Sept. '05, when it came on in spasms; vomiting from January onwards; latterly it ceased; weakness of legs; no teeth; has had syphilis.	Severe abdominal pains; iliac fossæ tender; vomited yesterday large amount of grey acid material, and felt relieved.		P. 128; T. 97.8; R. 28; abdomen fixed, distended, tender above umbilicus, over cæcum, and especially in left lumbar region; no supra-pubic tenderness; leucocytes 17,000.	Round ulcer, ⅝ inch, on lesser curvature extending backwards; much dark grey turbid fluid; ulcer very inaccessible; distended colon punctured to gain room and relieve tension; diaphragm and liver coated with lymph. Culture: pure bac. coli.	Ditto; peritoneal cavity washed out; wound and pouch of Douglas drained.	Pulse imperceptible; strychnine; P. 134, six hours later, and fell to 126 after transfusion of 1 quart saline; the respirations increased to 40, and he sank 14 hours after operation. Sectio: much plastic lymph about liver and diaphragm. Leakage from one suture.

3. OBSERVATIONS ON PERFORATED GASTRIC AND DUODENAL ULCER, BASED UPON A PERSONAL EXPERIENCE OF FORTY-SIX CASES OPERATED UPON

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These observations are based upon an experience of thirty-six cases of ulcer of the stomach, and ten cases of duodenal ulcer, on which I have had occasion to operate during the last few years on account of perforation or its immediate effects.

It is not necessary here to consider the general subject of gastric and duodenal ulceration, which for the most part lies within the domain of medicine rather than of surgery, although I need not remind this Society that these conditions no longer interest the physician alone. Discussions which have taken place within these walls have played no inconsiderable part in directing attention to the possibilities for good of surgical intervention in a limited, yet clearly defined, class of cases. If there is still room for discussion as to the precise rôle of surgery in the treatment of unperforated ulcers, there is, I think, no difference of opinion regarding its place when perforation has occurred. Attention will, therefore, be confined to the subject of perforated ulcers, chiefly in its clinical aspects, and, to avoid confusion, I shall refer to gastric and to duodenal perforations separately.

Sex Incidence of Perforation.—There is, I think, a general impression that not only does gastric ulcer occur with greater frequency in women than in men—the ratio, according to F. Brunner (2), who has analysed all the statistics published up to 1903, being as four women to one man—but that in the female there is a greater liability for the ulcers to perforation. It is undoubtedly true that women suffer from perforation oftener than men, but published statistics show that they do so only in so far as they are more frequently the subjects of ulcer.

My own series of cases—too small to justify any general conclusion—would seem to indicate that the liability to perforation is even greater in the male than in the female, for of 36 gastric cases operated upon, 25 were women and 11 men —a proportion roughly of 2 to 1. If I include my cases of duodenal ulcer, there are 20 males to 26 females (Table I.).

It would thus appear that men present symptoms of perforation almost as often as women, a fact which has a practical bearing on diagnosis, as there is a tendency to consider that the presumption is against a diagnosis of perforation of the stomach or duodenum when the patient is a male.

Age.—The youngest patient on whom I have operated for perforated *gastric* ulcer was a girl æt. 15½, and the oldest a woman æt. 55½. From the accompanying table (Table I.) it will be seen that perforation of gastric ulcers occurred twenty-two times in patients below the age of 30, and fourteen times in patients over that age. It will also be observed that below the age of 30 only three were males (nineteen females to three males), while above 30 the males predominate (eight males to six females).

TABLE I. AGE AND SEX.

A. *Perforated* GASTRIC *Ulcers*—36.

Age.	Females.	Males.	Total.
Between 15 and 20	10	2	12
„ 20 „ 30	9	1	10
„ 30 „ 40	4	5	9
„ 40 „ 50	0	3	3
„ 50 „ 60	2	0	2
	25	11	36

B. *Perforated* DUODENAL *Ulcers*—10.

Age.	Females.	Males.	Total.
Between 15 and 20	0	1	1
„ 20 „ 30	1	1	2
„ 30 „ 40	0	2	2
„ 40 „ 50	0	2	2
„ 50 „ 60	0	2	2
Over 60	0	1	1
	1	9	10

So far as I have been able to find, the youngest patient hitherto operated upon for perforation of a gastric ulcer was a boy, æt. 12, successfully operated upon by Mr Caird. The oldest patient of whom I can find a record was a man, æt. 71, also operated on by Mr Caird in 1905. The oldest patient who recovered was a man, æt. 62, operated upon by T. Myles of Dublin (5) in 1899. In this case the perforation occurred during the reduction of an umbilical hernia, and it is an interesting fact that Mr Caird's aged patient also suffered from umbilical hernia.

The age in the *duodenal* cases ranges from 19 to 64; the only female in the series was 21 years of age.

While in itself the age of the patient is not a factor of great importance in prognosis, it is interesting to observe that in the *gastric* series, of fourteen patients over 30 years of age, only four survived (Geo. O., æt. 31; Wm. L., æt. 34; Mrs K., æt. 36; and Mrs W., æt. 55½). Of the twenty-two patients under 30, fifteen recovered.

In the *duodenal* series, of the five patients who recovered, four were under the age of 40 and one was 52; and of the five who died, four were over 40 and one was 19 years of age.

It would appear, therefore, that, other things being equal, the younger the subjects the greater is the prospect of recovery.

Occupation.—There does not appear to be any traceable relationship between the occupation of the patient and the occurrence of perforation. All the patients led busy, active lives, the majority, however, being engaged indoors. In the *gastric* series, of the females eight were domestic servants, two shop-girls, six housewives, one matron of a charitable institution, one a hospital nurse, one a tailoress, one a clerkess, three factory-workers, one an upholstress, and one an out-door worker. The males included an insurance agent, a clerk, a baker, a vanman, and five labourers.

In the *duodenal* series, the female was a shop-girl, and the males included a teacher of mathematics, a cashier, a county sanitary inspector, a dispensing chemist, a grocer, a barman, two joiners, and a labourer.

Previous history of dyspepsia or of ulcer—Gastric Cases.—The great majority of my patients gave a history of having suffered for a longer or shorter period from indigestion, although

in a considerable proportion the symptoms had not been sufficiently severe to lead them to seek medical advice. One patient, for example, an insurance-agent, æt. 38, had been troubled for some years with gastric discomfort and griping pains after taking food, but although he suffered a good deal he had never consulted his doctor about the condition. There was no history of vomiting, hæmatemesis, or melæna. Perforation occurred one Saturday afternoon on the top of a tramway-car while he was returning from a game of golf.

In other cases the patients had been specially treated for dyspepsia, and in some were actually under treatment when the perforation occurred. This was so in the case of a hospital nurse, æt. 25, who for about three years had complained of a feeling of distension and some pain after food, so that she had to be very particular about her diet. There was no vomiting or melæna, or other symptom suggestive of gastric ulcer. Perforation occurred while she was engaged in decorating her ward for Christmas.

A. B. Mitchell (16) has met with two cases of perforation occurring in patients who were undergoing a course of treatment by rectal feeding.

In the *duodenal* cases, on the whole, the history of antecedent dyspepsia was more constant and more definite—a circumstance which may partly be explained by the fact that the patients were males. A history of discomfort or pain to the right of the umbilicus an hour and a half or two hours after food, of inability to take certain articles of diet, and of sickness, was not uncommon. One patient had vomited blood a year before perforation occurred; another had passed dark stools; and one had for some months been treated for dilated stomach by lavage, and had actually washed out his stomach as usual the night before perforation occurred.

In some of the *gastric* cases the patients had been free of symptoms of dyspepsia for a considerable period prior to the occurrence of perforation. This was the case, for example, in a clerkess, æt. 24, who for some years had suffered a great deal from indigestion, but after a course of treatment and careful dieting, had been apparently cured, and was for some months entirely free of discomfort and able to take ordinary diet with impunity. Perforation occurred one evening while she was walking home from business. A married woman, æt. 36, also

had enjoyed five years' complete immunity from symptoms of dyspepsia before perforation occurred. Schloffer (6) and Guinard (7) have reported similar cases.

In one of my *duodenal* patients also, this improvement in general health was very striking. He was a gentleman, æt. 50, who for years had suffered greatly from indigestion. For some months he had been less troubled, and after a prolonged holiday had returned to business feeling better than he had done for years, and able to take ordinary diet without discomfort. Without warning perforation occurred one night while he was in bed.

In a considerable number of cases the antecedent symptoms described were clearly those of ulcer of the stomach. With regard to the presence of a definite history of gastric ulcer, a striking difference between the sexes is manifest in my statistics, because while in only one man out of eleven was there a history suggestive of ulceration, in twelve out of twenty-five women the patients had either been treated for ulcer or the symptoms clearly pointed to the existence of such a condition. It is not improbable that previous evidence of ulcer is even more common than these figures would appear to indicate, as in several of the negative cases the patients when seen were too ill to bear cross-examination on such points, and the friends were often not in a position to give any information.

Only in five *gastric* cases—two men and three women—and two *duodenal* cases—one man and one woman—was there no history of previous gastric disturbance, the patients having taken ordinary diet without pain or discomfort, and apparently been in perfect health up to the moment of perforation. Pariser (1), Schuchardt (1), Cornil (1), Bourlot (1), Caird (4), and others have recorded similar cases.

These observations agree in the main with those of Mayo-Robson (8), who estimates that symptoms of ulcer may be latent in about 20 per cent. of cases, and only slight in others, but that in fully 50 per cent. there are marked symptoms. F. Brunner (2), from an analysis of 286 recorded cases of perforation, indicates that only $4\frac{1}{2}$ per cent. occur in patients apparently healthy, as against about 90 per cent. with a previous history suggestive of ulceration. Weir and Foote (9) calculate that in 92 per cent. there is an antecedent history of ulcer.

It is a curious fact that while a previous history of severe dyspepsia and even of ulceration is so very frequent, only in five gastric, and two duodenal cases was it definitely stated that the patient had at any time vomited blood. This absence of hæmatemesis from among the antecedent symptoms has also been observed by Mr Caird.

Premonitory signs of perforation—Gastric Cases.—It may be worthy of note that in a number of cases the symptoms of indigestion had become more pronounced for some time before the occurrence of perforation. For example, the nurse already referred to, who was a chronic dyspeptic, two days before perforation occurred had to go off duty and spend the day in bed on account of a severe attack of "indigestion" associated with pain in her left side. Next day she felt better and resumed her work, but the following day the ulcer perforated.

Another patient, an anæmic girl (Cath. F.), æt. 18, four days before perforation occurred had to go to bed for a day on account of unusually severe pain in the epigastrium after taking food. In a couple of days this had passed off, and she resumed her usual mode of living, until perforation took place.

In the case of another girl (Susan F.), æt. 20, who had been under treatment for dyspepsia for three months, for several days before her ulcer perforated the pain had become much worse, and the day before she had for the first time vomited a quantity of dark brown coffee-ground material. Another girl (Euph. L.), æt. 20, who had long suffered from indigestion, fourteen days before perforation took place, had an attack of what was diagnosed and treated as "acute gastritis," and during this attack she, also for the first time, vomited blood. A man (Danl. B.), æt. 36, who had suffered from indigestion for years, also had an aggravation of all his symptoms about a fortnight before his ulcer perforated; and in another man (Wm. O.), æt. 49, with a history of indigestion of three months' duration, there was a similar aggravation prior to the occurrence of perforation.

Duodenal Cases.—In several of my duodenal cases also there were premonitory signs. My only female patient, æt. 21, a week before perforation occurred had to go to bed for two days on account of sharp pain in her right side, but with no signs of gastric disturbance. She resumed her work and some

days later perforation occurred while she was engaged in her shop. A lad, æt. 19, felt ill with pain in his side the day before perforation occurred, but was able to go to work next morning. During the forenoon perforation occurred while he was working at his bench as a joiner. Two men had had no motion of the bowels for a week before perforation occurred.

Possible factors determining perforation.—I have carefully analysed the records of my cases to try to find some explanation of the final perforation of the ulcer into the peritoneal cavity, but I fear without any success. An inquiry regarding what the patient was doing when the symptoms indicative of perforation came on, for example, fails to reveal any constant factor which would account for the ulcer giving way. In the *gastric* cases in which this point was noted I find that—

5 were in bed,
4 were sitting by the fire,
3 were at table,
1 was sitting on a tram-car,
1 was driving on a baker's van,
1 was travelling in a train,
1 was stepping out of a train,
1 was walking upstairs,
7 were walking on the street,
1 was doing light house work,
1 was working in a factory,
1 was doing labourer's work,
1 was doing up her back hair,
1 was hanging clothes on a rope,
1 was decorating a Christmas tree.

The first fifteen, therefore, were practically at rest; the next nine were making some slight effort; and only the remaining six were engaged in such a way as to involve any muscular strain or stretching of the body.

In the *duodenal* cases, two of the patients were in bed when perforation took place, but one stated that he was turning round quickly when he was suddenly seized with intense pain, and he was definite in attributing its onset to his having over-stretched himself. Two were sitting by the fire; three were at work as shop-keepers, and one of them was reaching to take a box off

a high shelf when the pain came on ; one was at work as a joiner and one as a labourer. Only in the two cases mentioned was the patient aware of making any movement likely to stretch the body at the moment when the symptoms of perforation began, and in both of these the perforation was small and rounded, in no way suggestive of a tear.

Nor does a consideration of what the patient was doing when perforation occurred, viewed in relation to the size and shape of the aperture in the stomach, throw any clearer light on the point. It is true that in the case of the nurse who was engaged in decorating a Christmas tree, the perforation took the form of a longitudinal tear about five-eighths of an inch long in the base of the ulcer, and it is conceivable that this might be explained by stretching of the stomach in over-reaching herself, but a precisely similar condition was found in a girl (Euph. L.), who was walking on the street when her acute symptoms began. Further, in other patients who were similarly stretching themselves, in hanging out clothes and in dressing the hair, for example, the perforation took the form of a small, clean-cut aperture.

In this connection the case of a clerkess (B. H.), æt. 24, may be cited. She stated that fourteen days before the onset of her acute symptoms, while lifting a heavy roll of linoleum she felt a sharp pain just below the left costal margin, and that "an uneasy feeling" had persisted in this region, although in other respects she was perfectly well. At the operation we found a long, narrow tag of omentum firmly adherent to the upper edge of the indurated area of the stomach wall and just above the perforation. The position of the ulcer corresponded to the site of the sharp pain she had experienced a fortnight before, and it is possible that the pain which occurred while she was straining to lift the weight was due to traction exerted through this tag of omentum. This explanation of the pain is, of course, not reconcilable with Lennander's proposition that the wall of the stomach, the visceral peritoneum, and the omentum are alike insensitive to pain. If we accept Lennander's statements in their entirety, however, it is difficult to account for many of the painful manifestations of gastric and other abdominal conditions.

Another explanation of this case suggests itself, however, namely, that the muscular effort caused a slight tear in the

ulcer, and that the omentum had become adherent over the spot and so prevented leakage.

About two years ago I saw, with Dr James Mill, a patient, æt. 31, who had for some time previously suffered from symptoms of duodenal ulcer. Two days before I saw him, while helping to lift a heavy box, he was noticed to become "deadly white," and he looked so ill that his fellow-workmen sent for brandy and gave it to him. He had not been conscious of anything having given way at the moment, but a short time afterwards he was seized with intense pain in the pit of his stomach, doubling him up and causing him to cry out. On reaching his home he was in a state of profound shock, with a markedly sub-normal temperature, and a pulse of forty-five. There was pain all over the abdomen, and an area of great tenderness to the left of the epigastrium. He passed a very bad night, but next morning began to improve, and as the improvement was maintained he was not operated upon. The opinion we formed was that the muscular strain had determined rupture of a duodenal ulcer, which had set up a localised peritonitis. In the course of a few weeks he recovered completely.

Cases have been recorded in which perforation has occurred from such direct forms of traumatism as a blow on the side, a fall down a stair, and the attempt to reduce an umbilical hernia; also from muscular effort, such as lifting a heavy weight, and during attacks of coughing or vomiting, but it is remarkable how seldom, on the whole, any traumatic factor seems to come into play in determining perforation.

It might be expected that the ingestion of food should play a part in determining perforation, but my observations do not appear to bear this out. In the twenty-eight *gastric* cases in which the time of the last meal is noted, I find that perforation occurred—

in 3, while taking a meal,
„ 2, immediately after a meal,
„ 3, 1 hour „
„ 5, 2 hours „
„ 4, $3\frac{1}{2}$ „ „
„ 4, $4\frac{1}{2}$ „ „
„ 5, $5\frac{1}{2}$ „ „
„ 1, 6 „ „
„ 1, 8 „ „

In eight, therefore, the stomach was presumably full; in thirteen it probably still contained some food, and in seven it was presumably empty. In the majority of the cases in which the stomach was presumably full when perforation occurred, the patients were making no muscular effort at the moment, as most of them were either sitting at table or by the fire, or were in bed at the time. The condition of the stomach with regard to food, therefore, does not appear to have any important bearing on the occurrence of perforation.

In this connection, however, I should refer to a case in which a girl, æt. 16, who had never previously complained of any gastric disturbance, was suddenly seized with symptoms of perforation immediately after she had eaten a large quantity of raw oatmeal and then swallowed a jugful of cold water. On opening the abdomen a small, circular perforation was found in the centre of an indurated ulcer about the size of a shilling.

In the *duodenal* cases in which this point was noted, perforation occurred—

in 1, while taking a meal,
„ 1, 1½ hours after „
„ 3, 2 „ „
„ 1, 4 „ „
„ 2, 5 „ „
„ 1, 8½ „ „

From these facts no conclusions can be drawn.

Morbid Anatomy—Gastric Cases.—It is generally recognised that while ulcers occur with greatest frequency on the posterior wall of the stomach, the majority of those which end in perforation are situated on the anterior wall. This has been variously attributed to the greater mobility of the anterior wall, to the fact that it is less liable to form adhesions with adjacent structures, and to its greater exposure to traumatism.

F. Brunner (2) has collected the records of 320 cases of perforation, and of these 278 were on the anterior wall, and only 42 on the posterior—in other words, perforations on the anterior wall are about 6½ times more common than on the posterior.

In my own series 33 of the ulcers had perforated on the anterior wall, and only 3 on the posterior—anterior perforations therefore being eleven times more frequent than posterior. (Table II. A.)

Table II. Site.

A. *Perforated* Gastric *Ulcers.*

	Anterior Wall.	Posterior Wall.	Total.
Author's Cases . .	33	3 (1 in 11)	36
F. Brunner's Collected Cases . . .	278	42 (1 in 6.6)	320

	Cardiac End.	Body of Stomach.	Pyloric End.	Total.
Anterior Wall:—				
Author's Cases . .	4	12	17	33
F. Brunner's Collected Cases . . .	122	23	78	278
Posterior Wall:—				
Author's Cases . .	0	2	1	3
F. Brunner's Collected Cases . . .	14	10	7	42

B. *Perforated* Duodenal *Ulcers.*

(*All in* first *part of Duodenum.*)

	Anterior Wall.	Posterior Wall.	Total.
Author's Cases .	9	1	10

Brunner's statistics indicate that ulcers situated towards the cardiac end of the stomach more frequently perforate than those in the body of the organ, or at the pyloric end. These are his figures: *Anterior wall*—cardiac end, 122; body of stomach, 23; pyloric end, 78. *Posterior wall*—cardiac end, 14; body, 10; pyloric end, 7.

In my experience the reverse has been the case, as these figures show: *Anterior wall*—cardiac end, 4; body of stomach, 12; pyloric end, 17. *Posterior wall*—cardiac end, 0; body of stomach, 2; pyloric end, 1.

All observers seem to agree that the vast majority of perforations are met with towards the lesser curvature of the stomach.

Duodenal Cases.—In only one case of duodenal ulcer was the perforation on the posterior wall. It was that of a man, æt. 50, who had suffered from dyspepsia for years and had also been addicted to alcohol. One of the anterior perforations was close to the upper edge of the bowel, and one close to the lower edge. In one case, operated on sixteen days after perforation for localised abscess, from the situation of the pus the rupture was believed to have been on the anterior wall.

Multiple Perforations.—I have only once met with a case in which there was more than one perforating ulcer. It was that of a girl (Susan F.), æt. 20, who was sent into Leith Hospital in June 1904 by Dr Wm. Paterson. She had suffered from anæmia and symptoms of gastric ulcer for about three months, and during the latter half of that time had been under treatment. Perforation occurred during the middle of the night while she was asleep, but the doctor was not sent for till late next forenoon. By the time she reached hospital she was *in extremis*, and some time was lost in obtaining the consent of the parents to operation. The abdomen was opened fifteen hours after perforation took place. An aperture, which was large enough to admit the tip of the index finger, was found in the anterior wall of the body of the stomach, close to the lesser curvature. It occupied the centre of an ulcer about the size of a half-crown piece. The perforation was closed, the abdomen washed out, and the patient put back to bed. She died a few hours later, and after death it was found that there was a second ulcer on the corresponding part of the posterior wall which was just on the point of perforating.

This is by no means an isolated observation. Mikulicz (10), Helferich (1), Barling (11), and others have reported similar cases. Demons (12) and Stelzner (13) have each sutured a perforation on the anterior wall, and at the autopsy found a second perforated ulcer on the posterior wall. Brunner has met with no fewer than thirty-nine cases of multiple perforations in 120 autopsies.

Size of Ulcers and Perforations — Gastric Cases. — The ulcers I have met with have all been rounded or oval, and their size, as estimated by the area of induration in the stomach

wall, has varied from that of a shilling to that of a crown-piece. In some cases the thickening and induration were comparatively slight, in others they were so marked as to give to the area of stomach wall implicated almost a cartilaginous consistence. In one instance the tissue surrounding the perforation was nearly three-quarters of an inch in thickness, and so dense that it was necessary to excise the whole ulcer before the aperture in the stomach could be closed.

In the great majority the perforation took the form of a small rounded or oval hole, with regular, clean-cut edges, as if a piece had been punched out of the centre of the ulcer. In most cases the opening just admitted the point of a pair of Kocher's artery forceps (2 mm.); in some it admitted the forceps up to the joint (8 mm.).

In five cases the perforation was so small that only the tip of a fine silver probe could be passed through it; while in four others the opening was as large as a threepenny-piece, admitting the tip of the little finger. Two of these large perforations were on the posterior wall of the stomach, and in neither was the ulcer of large size or particularly thick, and there were no adhesions related to it. Another was on the lesser curvature, and the ulcer involved both the anterior and posterior walls of the stomach. It may be remarked that each of the three perforations which I have met with on the posterior wall was larger than any of those occurring on the anterior wall.

It is interesting to note in passing that while all the five patients with minute perforations recovered, the four with large perforations succumbed. In the duodenal cases, also, it was observed that recovery took place in all the cases in which the perforation was minute, while the larger perforations proved fatal.

In two cases the perforation took the form of a longitudinal tear, $\frac{5}{8}$ in. and $\frac{3}{4}$ in. long respectively, and in each the ulcer was exceptionally thick and indurated. In one of these patients there was a distinct history that perforation followed during or soon after muscular effort, but in the other the patient was walking quietly on the street when the perforation occurred, and was not aware of having unduly exerted herself in any way.

In the *duodenal* cases the ulcers varied in size from about a quarter of an inch to an inch in diameter, in some cases

being thick and indurated, in others comparatively thin and soft. Five of the perforations were minute, admitting only the end of a probe; four were larger, varying in diameter from an eighth to a quarter of an inch.

Relation to Adhesions. — In the great majority of cases, both gastric and duodenal, there were no organised adhesions in the vicinity of the ulcer which had perforated.

In two cases of perforation of the anterior wall, a portion of omentum was firmly adherent to the stomach over the area occupied by the ulcer, and in each the patient had complained of sharp pain below the left costal margin for about two weeks before perforation occurred. It is possible that this pain was due to traction on the adherent omentum, and this view receives support from the fact that one of the patients volunteered the statement before operation that the pain was at once relieved when she took off her corset and lay down in bed.

In other two cases there were adhesions between the liver and the lesser curvature of the stomach, and in one of these pain had been a prominent symptom for some time before perforation occurred.

In one case there was a long adhesion between the great omentum and the anterior abdominal wall, which shut off the upper segment of the peritoneal cavity across the greater part of the abdomen. At each side, however, there was an open channel which permitted of the diffusion of fluid when perforation occurred.

Clinical Features.

It is important to discriminate clearly between the symptoms which come on immediately on the giving way of the ulcer and those which ensue later as a result of infection of the peritoneal cavity. The former present certain features which are characteristic of perforation of the stomach or duodenum, and are helpful in diagnosis; the latter are common to nearly all forms of acute infective peritonitis, and are therefore only of diagnostic value in the light of what preceded them.

Pain. — The most striking, constant, and characteristic symptom of the occurrence of perforation is the sudden onset of intense abdominal pain, which is variously described by different patients as of a stabbing, cutting, tearing, burning, or twisting character. It is always of extreme severity,

doubling the patient up, causing him to writhe in agony, and usually to cry out. The initial pain, as a rule, radiates all over the abdomen, and the patient indicates its site by sweeping the hand across the belly or round the umbilical region. Only in two of my cases have I been able definitely to elicit a history of pain referred through to the back, between the shoulders. I have not observed that radiation of pain towards the clavicular region to which Adamson and Renton (14) have drawn attention. In most cases there is an area situated in the epigastrium, or in one or other hypochondrium, in which the pain reaches its maximum intensity.

In a few instances, both in gastric and duodenal cases, the pain is specially referred to the right iliac fossa, and this may lead to an erroneous diagnosis of appendicitis. I have twice been misled in this way, and have cut down over the cæcum only to find the appendix normal. In one of these cases, that of a man (John B.) aged 20, the patient was so definite in his statement that the pain was, and always had been, most severe in the right iliac fossa, and the tenderness was so much more marked there than elsewhere, that I revised my original diagnosis and exposed the appendix.

In most cases, after having lasted for a period varying from half an hour to about two hours, this overpowering pain gradually eases off, and the patient experiences a sense of relief, tempered by the feeling that at any moment the suffering may be renewed.

The whole abdomen remains tender to touch, and by careful palpation an area in which the tenderness is most marked can generally be delimited, and this is usually found to correspond with the position in which the initial pain was most severe. In the majority of cases even light pressure cannot be borne over the abdomen, the muscles of which are tightly braced up, but several of my patients stated that bending forward and compressing the abdomen relieved their pain.

Apart from the general tenderness of the abdomen, I have not observed the excessive tenderness of the supra-pubic region referred to by Mr Caird.

I have found a remarkable correspondence between the site of the maximum tenderness and the seat of the perforation. When the most tender area was in the left hypochondrium, the perforation has almost invariably been towards the cardiac end

of the stomach ; when around the umbilicus, in the body of the stomach ; and when in the right hypochondrium, near the pyloric end or in the duodenum. So constant is this association that I have come to rely upon it as a guide to the site of incision.

Vomiting.—Vomiting is by no means a constant symptom at the onset of perforation. In eight of my *gastric* cases I have no note regarding this point. Of the remaining twenty-eight, ten vomited and eighteen did not. In several of these last, however, the patient felt sick, and in one or two others there was retching but no actual vomiting. When present, the vomiting was neither profuse nor persistent in any of the cases.

There appears to be considerable variation in the experience of different observers with regard to vomiting at the onset. Thus Roux (1), Dreschfield, Jaboulay, and Viannay (15), consider it a rare symptom ; Brunner (2) estimates that it occurs in about a third of the cases ; while Broadbent, Caird, and Stawell have found it commonly present.

In the *duodenal* cases in which I have a note on this point, only two vomited, and in both the sickness persisted and was accompanied by hiccough ; one recovered and one died. Seven of the patients did not vomit.

Abdominal Rigidity. — Rigidity with retraction of the abdominal walls is one of the earliest and most constant symptoms. Usually within a very few minutes of the onset of pain, the reflex or protective contraction of the parietal muscles and of the diaphragm renders the walls so tense that it is impossible to palpate the abdominal contents, and the movements of respiration are carried out entirely by the thoracic muscles. While rigidity is a symptom common to many acute abdominal conditions associated with infection of the peritoneum, it occurs earlier, I think, more rapidly becomes general, and is more pronounced in cases of gastric or duodenal perforation than in almost any others. Where much free gas is present in the peritoneal cavity, there is sometimes ballooning of the abdomen.

With the advance of peritonitis, the rigidity becomes even more marked, but the retraction gives place to distension, which may be due to accumulation of free gas in the peritoneal cavity or to tympanitic distension of the intestines.

In one of my cases, four hours after perforation had occurred, and in another, thirty-six hours after, the lower part of the abdomen continued to move with respiration ; and in a third, at the same period, the abdominal rigidity had entirely disappeared and the abdomen was quite flaccid. The latter patient was suffering from a marked degree of toxæmia, and her condition was complicated by the presence of a right-sided pneumonia of which she died on the fifth day after operation. D'Arcy Power (17) and Poucet (18) have recorded similar cases.

Percussion.—The information obtained by percussion varies widely in different cases, and its value is not always easy to estimate. In many cases the muscular rigidity and the extreme tenderness of the abdomen preclude the possibility of employing percussion.

In early cases, when there is much free gas in the general peritoneal cavity, a characteristically high-pitched, drummy note may be elicited all over the abdomen, so that not only is it impossible to differentiate the hollow viscera from one another, but percussion over the solid viscera yields the same note.

When there is an abundant effusion of peritoneal fluid, the dependent parts of the abdomen may yield a dull note on percussion.

In one of my cases, that of a young woman (E. B.) æt. 30, a well-defined area of dulness could be recognised extending from the left hypochondrium, through the umbilical region, to the left iliac region, and there was marked tenderness on making pressure over this area. The shape and disposition of this dull patch led us to believe that perforation had taken place on the posterior wall of the stomach, and that there was an accumulation of fluid in the lesser peritoneal sac. On opening the abdomen the lesser sac was found distended, and on making an opening through the anterior layer of the gastro-colic omentum, a large quantity of turbid, yellow fluid escaped. This fluid could be pressed through the foramen of Winslow into the greater sac. The perforation on the posterior wall having been sutured, both peritoneal sacs were washed out and drained, and the patient recovered.

In another girl (E. L.), æt. 20, the area above the transverse colon, including the hepatic area, yielded a high-pitched drummy note suggestive of free gas in the peritoneal cavity,

while the part below the colon yielded a normal intestinal note. On opening the abdomen free gas was found in the cavity, but no explanation of its limitation to the area above the colon was forthcoming.

Liver dulness.—Considerable importance has been attached by some writers to the loss or diminution of the normal liver dulness in the diagnosis of perforation, but I have found this a sign which it is often difficult to estimate with certainty and to assess with accuracy. Even when, as has happened in a majority of my cases, the area of liver dulness is entirely absent or appreciably diminished, it is not always easy to determine whether this is due to the presence of free gas in the peritoneal cavity, to distension of the intestine, or to displacement of the liver by distended bowel.

When, very soon after the onset of pain, the hepatic area yields a high-pitched note, the presumption is that gas has escaped from a perforation; but when several hours have elapsed the other possible explanations must be taken into account. After twenty-four hours, the absence of liver dulness has no diagnostic value.

In my experience the liver dulness has been absent more frequently in cases seen a considerable time after perforation than in those seen early, but I cannot trace any constant connection between the time which has elapsed and the absence of the dulness. It was completely lost, for example, in two cases seen within two hours of perforation; while it was still present in other cases in which 23, 36, and 43½ hours had elapsed.

In the *duodenal* cases the liver dulness was lost in patients operated on respectively, 15, 20, 56, and 60 hours after perforation; and was present in others operated on 3½, 11½, 18½, and 50 hours after.

Nor have I been able to find any connection between the site of the ulcer and the presence or absence of liver dulness. Perforations at all parts of the anterior wall of the stomach have been associated with loss of liver dulness, others with its presence, and even when the perforation has been on the posterior wall, this symptom has varied, as in one case the dulness was completely lost, while in another, almost parallel, case, it was unimpaired.

Even the presence of free gas in the peritoneal cavity does

not seem to determine a loss of the hepatic dulness, as in several cases the liver area was dull although a large quantity of gas escaped when the peritoneum was opened.

It would appear, therefore, that while an early absence, or marked impairment, of the liver dulness is suggestive of perforation, its disappearance later is of little diagnostic significance, while the persistence of the normal dull area in no way negatives a diagnosis of perforation.

Initial Shock.—In all cases the onset of the pain which marks the occurrence of perforation is rapidly followed by the development of a condition of profound shock. The pulse becomes small, feeble, and rapid (100-120); the temperature falls below normal; the respiration becomes shallow, rapid, and sighing, and owing to the rigidity of the abdominal muscles it is entirely thoracic; the face is pale and drawn; the extremities become cold and clammy.

Period of Reaction.—In a considerable number of cases, in my experience in the majority, the initial shock gradually passes off, the severity of the pain abates, and after a time the patient both looks and feels much better. The period within which this reaction takes place varies from one to four hours, the improvement sometimes taking place rapidly, sometimes more gradually. In several cases, although the patients had been very ill at the onset, the improvement which accompanied reaction was so great that the friends did not at once send for the doctor, and he was only summoned some hours later when symptoms of peritonitis developed. So marked may the improvement be that it is sometimes difficult to persuade the patient or the friends that the condition is a serious one. The disappearance of the symptoms may even lead the medical attendant to revise his diagnosis. I have in my mind at least one case in which the doctor in attendance diagnosed perforation within two hours of its occurrence, and arranged for the patient being admitted to hospital; but, on his return in an hour to accompany her in the ambulance waggon, he found her so much better that she was not sent in. A few hours later, symptoms of general peritonitis developed, and, although the patient was eventually operated upon, she succumbed. In another case, in which perforation occurred on the street, the patient was taken direct to hospital, and admitted to a medical ward. From the notes there made I find that the original

diagnosis of perforated gastric ulcer was set aside, in view of the marked improvement which took place within a couple of hours, and as this improvement was maintained for nearly three hours the patient was on the point of being sent out, when symptoms reappeared somewhat suddenly and she was at once transferred to the surgical wards. Recovery followed the operation in this case.

It is to be observed, however, that in spite of the general improvement which accompanies this temporary reaction, the pulse becomes progressively more rapid and loses in quality, and this should serve to prevent us being misled. The importance of clearly recognising the nature of this apparent improvement in the patient's condition is so evident that I need not labour the point.

In some cases there is no distinct reaction from the initial shock, the symptoms of general peritonitis gradually supervene, and the patient passes into a condition of collapse.

I should like here to refer to the question of *the administration of opium* for the relief of the pain which accompanies perforation. It must at once be admitted that it is much easier to declaim *ex cathedrâ* on the evils of opium in acute abdominal conditions, than to withhold the drug when face to face with a patient in the agonies which attend many of these conditions. To anxious and sympathetic relatives the hackneyed plea that opium "merely masks symptoms" is unconvincing, particularly when all they ask at the moment is that symptoms should be masked.

I shall not here refer to the vexed question of opium in abdominal surgery, further than to say that my statistics regarding its use in cases of perforated gastric and duodenal ulcer clearly indicate that it is attended with very great danger. In 34 of my records I have a definite statement regarding opium. In 22 of the cases no opium was given; 5 of the patients died, and 17 recovered. In 12 in which opium was given, 10 died and only two recovered, and in both of those in which recovery took place a definite diagnosis was arrived at, and preparations made for operation, before the opium was administered.

The accompanying table (Table III.) shows the period which elapsed between the occurrence of perforation and the performance of the operation in these cases, and it will be seen

that the time factor exercised no more than its usual influence in determining the result. Doubtless a considerable time elapsed in some of the fatal cases, but it is fair to assume that had the patient not been under the influence of opium this period would have been materially curtailed.

It may be a counsel of perfection, but one cannot avoid the inference that in cases of suspected perforation opium should be withheld.

TABLE III.

A. *Perforated* GASTRIC *Ulcer.*

Cases in which NO OPIUM *was administered*—17.

No.	RECOVERY. Interval between Perforation and Operation.	No.	FATAL. Interval between Perforation and Operation.
1.	4 hours	1.	4 hours
2.	$4\frac{1}{4}$ „	2.	15 „
3.	6 „	3.	18 „
4.	$6\frac{1}{2}$ „	4.	36 „
5.	10 „		
6.	11 „		
7.	$11\frac{1}{2}$ „		
8.	15 „		
9.	21 „		
10.	23 „		
11.	35 „		
12.	36 „		
13.	36 „		

Cases in which OPIUM *was administered*—9.

No.	RECOVERY. Interval between Perforation and Operation.	No.	FATAL. Interval between Perforation and Operation.
1.	$1\frac{3}{4}$ hours	1.	3 hours
2.	8 „	2.	5 „
		3.	10 „
		4.	17 „
		5.	25 „
		6.	$43\frac{1}{2}$ „
		7.	96 „

B. *Perforated* DUODENAL *Ulcer.*

Cases in which NO OPIUM *was administered.*

RECOVERY.		FATAL.	
No.	Interval between Perforation and Operation.	No.	Interval between Perforation and Operation.
1.	2 hours.	1.	56 hours.
2.	3½ „		
3.	15 „		
4.	20 „		

Cases in which OPIUM *was administered.*

RECOVERY.		FATAL.	
No.	Interval between Perforation and Operation.	No.	Interval between Perforation and Operation.
		1.	11½ hours.
		2.	18½ „
		3.	2½ days.

Onset of Symptoms of Peritonitis.—Sooner or later the apparent improvement which accompanies the reaction from the initial shock ceases and symptoms of infection of the peritoneum supervene. When the perforation is of some size and situated in the anterior wall of the stomach, and when there are no limiting adhesions, these symptoms are referable to the greater sac of the peritoneal cavity, are early of onset and rapid in their progress, so that within a few hours the patient has passed into a condition of profound collapse.

Perforation of the posterior wall of the stomach leads to primary infection of the lesser sac, but if the foramen of Winslow is patent, the infective material soon reaches the greater sac. If the foramen has been occluded by adhesions, however, the infection may remain confined to the lesser sac and eventually result in the formation of a localised abscess.

There is great variation in the time of onset, rapidity of progress, and extent of spread of the peritonitis. Two of my patients were moribund within eighteen hours of perforation, and of the others on whom I operated when *in extremis*, the time since perforation varied from twenty-five hours to four days. In one exceptional case perforation was believed to have occurred nine days before.

An analysis of my cases has not enabled me to determine the factors upon which the progress of the peritonitis depends. I am unable to confirm the generally accepted opinion that the acuteness of the infection is influenced by the size of the perforation, its situation, the quantity or character of the material which escapes through it, or the state of repletion of the stomach at the moment when perforation occurs. Doubtless each of these factors plays a part, but in addition such circumstances as the general condition of the patient, whether or not she was moved or has taken food or medicine after the ulcer gave way, and others, have to be taken into account.

The clinical features of the peritonitis which supervenes are similar to those of other forms of perforation peritonitis, and need not be described here. Dudgeon and Sargent (19) have isolated from the edge of all the gastric ulcers they have examined, and from the exudate in a majority of their cases, a streptodiplococcus, as yet unclassified, of low pathogenic power in the human subject. They quote Ghon, Jensen, and Brunner as having frequently found the pneumococcus present.

TREATMENT.—*Gastric Ulcer.*—I know of no qualification to the statement that the only treatment of perforated gastric ulcer is to open the abdomen, close the aperture in the stomach wall, and cleanse the peritoneal cavity. This should be done with the least possible delay, and however advanced the condition may be, the patient should never be denied the chance which an operation affords.

Incision.—It has already been pointed out that the seat of maximum pain and tenderness nearly always corresponds to the site of the perforation, and over this area the incision should be made. Personally, I prefer a vertical incision, on one or other side of the middle line, passing between the fibres of the rectus muscle. I have employed transverse and ⊣ shaped incisions, but these are only to be made when access to the area of perforation cannot be obtained through a vertical incision. When the patient is too ill to appreciate degrees of pain or to localise the most tender area, the incision most generally useful is one carried through the inner third of the left rectus. A short incision should first be made to verify the diagnosis. When perforation has occurred there is often considerable œdema of the extra-peritoneal connective tissue, and when free gas is present in the abdominal cavity

the parietal peritoneum usually bulges out into the wound. On dividing the peritoneum the gas escapes with a whistling sound, and often the distended abdominal parietes collapse and become quite flaccid. Seldom has the gas any distinctive odour; but occasionally, in cases of advanced peritonitis, the odour characteristic of the *bacillus coli* may be detected.

As soon as the diagnosis is thus confirmed, the incision should be enlarged to the extent of 5 inches in order to expose thoroughly the region of the perforation. As this is being done, a quantity of fluid, mixed with gas, usually comes bubbling out through the wound. The amount of fluid varies from a few ounces to several pints, but the quantity does not seem to bear any constant relation to the site, size, or character of the perforation, to the previous condition of the stomach with regard to food, nor even to the time which has elapsed since the ulcer gave way. It is true that, as a rule, the quantity is greater in cases in which a considerable time has elapsed, but in some of my earliest cases—operated on within two hours of perforation—several pints of fluid escaped, and the fluid was already distributed all over the abdomen, the pouch of Douglas containing the greater bulk of it.

The fluid varies in character in different cases, sometimes being thin, pale yellow, and slightly turbid, sometimes ochre-coloured and containing flakes of greyish-white lymph, sometimes brownish as if from admixture with blood. In some of the early *duodenal* cases it has been milky-white, and in others glairy and of a deep orange colour from the presence of bile. In a number of cases the fluid had a distinctly sour smell.

It is remarkable how seldom the fluid contains any recognisable food particles, even in cases where the perforation occurred soon after a meal. The only food particles of a solid nature I have been able to identify have been flakes of curdled milk. In three cases I have recognised castor oil, which had been taken by the patient after the onset of symptoms, and in one case the fluid which escaped smelt strongly of whisky, which also had been taken subsequent to perforation. I cannot avoid the conclusion that in the great majority of cases the stomach contents do not escape in any quantity into the peritoneal cavity.

Finding the Perforation.—As a rule, there is little difficulty

in finding the perforation. The main obstacle is usually the presence of fluid, which fills up the wound and obscures the stomach. If this be swabled up with gauze or washed away with a stream of saline solution, and the wound then well dried, it will generally be noticed that with each respiratory movement of the abdominal wall more fluid mixed with air comes gurgling up from one region, and here the perforation will be found. Sometimes the position of the aperture may be recognised by the presence of a layer of newly formed grey lymph adherent to the stomach wall, or by recent adhesions between the omentum and the stomach, and it may even be necessary to separate these to expose the opening. If these guides fail, the anterior wall of the stomach should be systematically examined, passing from the pylorus towards the cardia along the lesser curvature, and then back along the greater curvature to the duodenum, which is in turn carefully investigated. If no perforation is thus found, the gastro-colic omentum should be torn through and the posterior wall examined in the same way. I have never had occasion to open the stomach and explore it from the inside, nor do I think this can often be necessary. From what I have seen, I am inclined to believe that if there is no evidence of an ulcer or of perforation on the surface of the stomach, the diagnosis has probably been wrong, and no good, but rather harm, is likely to follow opening the stomach.

The various forms of perforation met with have already been described. The aperture is generally situated near the centre of an indurated patch in the stomach wall, which marks the site of the ulcer, and when it is exposed a small quantity of pale, glairy mucus mixed with gas is usually seen bubbling from it. In some cases the mucus is bile-stained, particularly in duodenal perforations.

Closure of the Perforation.—As soon as the perforation is found, a small plug of iodoform gauze should be packed into it to prevent further escape of the stomach contents. If possible, the portion of the stomach on which the ulcer is situated should then be pulled well out of the wound and packed round with sterilised gauze wrung out of hot saline solution.

Should the stomach contain much fluid or be distended with gas, which is not often the case, it may be emptied by means of a catheter passed through the perforation. In several

cases I have washed out the stomach in this way before proceeding to close the perforation.

In the majority of cases it is possible to occlude the perforation completely by an ordinary Czerny-Lembert suture, or by a double row of Lembert sutures, of fine intestinal silk. Over these stitches, an omental graft, which can almost always be readily obtained, should be fixed, to add to the security of the closure. This method I have found, on the whole, preferable to the purse-string suture, which, on account of the density of the area surrounding the perforation, often fails to pucker up the wall sufficiently to occlude the aperture. In any case, a row of Lembert sutures and an omental graft should be employed over the purse-string suture.

As might be expected from the greater depth of the duodenum, and from its fixation, it is usually more difficult to ensure perfect closure of duodenal perforations than of those in the stomach.

Only in two of my cases have I excised the ulcer. In one, it was found impossible to invaginate the edges of the perforation, which took the form of a slit about $\frac{5}{8}$ inch long. The difficulty arose from the fact that the ulcer, which was about the size of a five-shilling piece and encroached on the lesser curvature and posterior wall of the stomach, was nearly $\frac{3}{4}$ of an inch thick and as dense as cartilage. The edges of the gap left after excision of the ulcer were brought together by Czerny-Lembert sutures, and a tag of omentum was grafted over the line of the stitches. The patient died forty-six hours after operation. In the second case, that of a man, æt. 49, on whom I operated when he was *in extremis*, forty-eight hours after perforation had occurred, a thick, indurated ulcer was found close to the pylorus, and it was evident that any attempt to invaginate the edges of the perforation would result in occlusion of the pyloric orifice. Even after the ulcer had been excised and the edges of the gap brought together, the pylorus was so constricted that I hesitated to return it, and performed a posterior gastro-enterostomy. This patient only survived the operation nine hours.

In the case of a man, æt. 50, I found that closure of a duodenal perforation narrowed the bowel to such an extent that there was danger of obstruction. I therefore performed a posterior gastro-enterostomy, but without success.

In another case, that of Mr K., æt. 36, operated on twenty-

one hours after perforation, the ulcer was situated so close to the pylorus that any attempt to invaginate the edges would inevitably have occluded the pyloric orifice. As the patient was too ill to justify prolonging the operation by performing gastro-enterostomy, I slit up the ulcer longitudinally, and, opening out the gap, stitched it at right angles to the long axis of the pylorus, thus performing pyloroplasty. Over the line of sutures I grafted a portion of omentum. This patient recovered.

The same procedure was carried out in a duodenal case, in a man, æt. 19, fifty-six hours after perforation, but the patient did not recover.

If one might venture to express a general opinion on such limited data, one would suggest: (1) that excision of the ulcer is seldom necessary to secure occlusion of the perforation, and, unless absolutely essential for this purpose, it is not to be recommended; (2) in comparatively few cases is the patient in a condition to justify gastro-enterostomy being performed merely as a precautionary measure against stenosis of the pylorus, or as an additional means of favouring healing of the ulcer, although this operation may be necessary in cases where the closure of the perforation has so encroached on the pyloric orifice that there is a danger of obstruction to the onward flow of the stomach contents; (3) in certain cases pyloroplasty is a simple and efficient alternative to gastro-enterostomy.

Purification of the peritoneal cavity.—Only in one case have I relied upon swabbing with gauze alone for the cleansing of the peritoneum. In this case the perforation was a very minute one, and, although six and a half hours had elapsed, the effused fluid was confined to the region of the stomach by recent adhesions. The means adopted was effectual, and the patient recovered.

In three gastric cases and in one duodenal case, the patients being *in extremis*, after closing the perforation and mopping up the excess of fluid with gauze, I have been content to pass large glass tubes filled with iodoform worsted in various directions. This is not a method to be recommended, and it has only been adopted when it was feared that the patient would succumb on the table if more were attempted. These four cases were fatal.

In all my other cases I have freely irrigated the peritoneal cavity with normal saline solution or boiled water. I have invariably made a supra-pubic opening to obtain access to the pelvis, and in almost every case, even the earliest, have found a considerable quantity of turbid yellow fluid in the pouch of Douglas. The apparatus employed is a glass uterine douche tube attached to four feet of rubber tubing, and a small glass filler. Large Keith's tubes are passed into the peritoneal cavity in various directions, so that the fluid may have free exit, and every corner of the abdomen is systematically washed out. The region of the perforation is usually flushed out first, and next the pouch of Douglas through the supra-pubic opening made for the purpose. The other parts of the abdomen are then systematically explored in the following order: (1) the left sub-phrenic space; (2) the splenic region; (3) the left iliac fossa; (4) the right sub-phrenic space; (5) the region of the hepatic flexure; (6) the right iliac fossa. Before closing the upper wound, the pouch of Douglas is once more flushed out.

Drainage.—After repeated trials, I have given up draining the region of the perforation, as I have seldom found that anything comes by the tubes left here. If there is doubt as to the security of the stitches in the stomach wall, a cigarette drain of iodoform worsted is passed down to the region of the perforation, and the rest of the wound is closed. This is most often necessary in duodenal cases, on account of the greater difficulty in suturing the perforation, and owing to the more definite and localised area of infection it is more frequently efficient. The pouch of Douglas is always drained by means of a large glass tube, similar to a Keith's tube, but without the lateral openings, into which tags of omentum are liable to pass, blocking the apertures and preventing the tube being withdrawn. These omental tags are sometimes so firmly fixed that it is necessary to cut them off on the outer side of the tube. To improve the drainage through the glass tube, a few strands of iodoform worsted, preferably in the form of a cigarette drain, should be inserted into it. In most cases the tube may be removed on the second or third day.

I am fully convinced of the value of raising the head of the bed on blocks six or eight inches high, as soon as the

patient has recovered from the shock of the operation. I have done this ever since the late Ryerston Fowler first suggested it, and have found it of the greatest advantage in all cases of general infection of the peritoneum.

Raising the head of the bed on blocks is preferable to propping the patient up with pillows, as the declivity of the pelvis can be maintained constantly and without effort on the part of the patient.

After-treatment.—Saline injections—15 to 20 oz.—are given by the rectum every hour, or every two hours. In some cases a continuous stream of warm saline solution has been allowed to flow slowly into the rectum from a douche can placed above the head of the bed. In several cases, where the shock was severe, and the signs of toxæmia marked, great benefit has followed direct intra-venous infusion of saline solution, the cannula being left in position in the vein, and the injections repeated every two hours.

The patient is allowed to wash out the mouth with tepid water from the first, but not to swallow anything for twenty-four hours. After this, sips of warm water or of albumen water are given frequently, and after forty-eight hours peptonised milk, beef juice, beef jelly, etc., is given in small quantities. By the end of the first week the patient is having milk food, gruel, porridge, rice soup, etc.

I cannot avoid feeling that the withholding of food by the mouth in these cases is often over-done, and that the fear of leakage through the perforation is exaggerated. After the operation the stomach is not a dry, empty sac, but contains a considerable quantity of mucus and gastric juice, which is just as likely to leak through our stitches as any fluid we may introduce.

A purgative, four grains of calomel, or half an ounce of castor oil, is usually given on the evening of the fourth day, and is followed by an olive oil or turpentine enema next morning.

The drainage tube is removed on the second or third day.

I have noticed that, while the epigastric wound usually heals by primary union, in quite a number of cases a small collection of sero-purulent matter forms from the seventh to the tenth day, and requires to be let out. This is not asso-

ciated with any pyrexia or other sign of absorption, and the small opening rapidly closes.

Complications.—Only in one case has a *gastric fistula* formed. It was in a girl (A. T.), æt. 20. The perforation was situated at the pylorus, near the lesser curvature, and the ulcerated area was densely indurated and very difficult to invaginate. As it was felt that the sutures were insecure a rubber tube surrounded with iodoform worsted was passed down to the region of the perforation. On the third day there was an escape of stomach contents, including some unaltered currants which the patient had eaten two days before perforation occurred. The fistula discharged mucus, gastric juice, and food particles for nearly a week, and then gradually closed. On the ninth day after the operation this patient developed *pneumonia*, which lasted for six days, the temperature ranging from 102° to 104° F., the pulse from 126 to 140, and the respirations from 40 to 52. A week after having her crisis, she developed an *acute parotitis* on the right side, with a rise of temperature to 101° F. In the course of a few days this subsided without going on to suppuration, and the patient eventually recovered.

A girl (I. M'L.), æt. 16, died of *pneumonia* on the fifth day. Another girl (C. F.), æt. 18, had a rise of temperature to 101.4° F., on the fourth day, which was the beginning of a sharp attack of *bronchitis*, which lasted for four days and then gradually passed off.

A woman, æt. 55, some weeks after operation developed a large *pelvic abscess*, which was opened and eventually healed.

The recovery of another patient, Emily B., æt. 30, was complicated by an attack of *phlebitis* in the left leg, which began sixteen days after operation, with severe pain and swelling in the calf, attended with rise of temperature (101.8° F.), and lasted for nine days. In this case the perforation was on the posterior wall of the stomach, and there was an effusion into the lesser sac.

Prognosis.—The only factor which appears to have any determinable influence on the prognosis is the period which is allowed to elapse between the occurrence of perforation and the performance of operation. While it is true that early operation does not in all cases avert a fatal issue—I have lost patients operated upon as early as two, three, and five hours after perforation—statistical evidence all

goes to prove that every hour of delay diminishes the patient's chance of recovery. Table IV. shows the interval which elapsed between perforation and operation in my cases. This point is so evident and so generally recognised that it need not be dwelt upon.

I have analysed my records to see if any other factor can be traced which appreciably influences the prognosis.

It might be expected, for example, that in *gastric* cases the *condition of the stomach with regard to food* would be of some importance in this connection, and that the prognosis would be less favourable if the stomach were full when perforation occurred. Statements to this effect are frequently made by writers on the subject, but I find no support for them in my experience.

Thus, two of my patients were actually taking a meal when perforation occurred, one vomited everything she had taken, but the other did not vomit. Both recovered. Of two, in whom perforation occurred immediately after a meal, one recovered and the other died, and both had vomited at the onset of the symptoms of perforation. There were three cases in which perforation occurred one hour after a meal; two of the patients died and one recovered. Both of those who died had vomited at the onset. I have no note regarding vomiting in the one who recovered. Of five patients in whom perforation occurred two hours after a meal, four recovered and one died, and in only one of those who recovered had there been vomiting.

Of twelve *gastric* cases, therefore, in which rupture took place within two hours of a meal, and in which presumably the stomach contained a considerable quantity of food, seven recovered and five died, and of those who recovered the majority had not emptied the stomach by vomiting.

If we note the time which elapsed between perforation and operation in those cases which recovered, it becomes evident that the issue was determined by early operation rather than by the condition of the stomach with regard to food, because in five of the seven the operation was performed respectively within two, four, eleven, eleven and a half, and fifteen hours of perforation; in the other two the interval was considerably longer—thirty-five and thirty-six hours respectively. In four of the five fatal cases, on the other hand, the interval was over seventeen hours,

namely, seventeen, twenty-seven, thirty-five, and thirty-six hours, and in one it was only five hours.

In the *duodenal* cases it is equally evident that the interval between perforation and operation was the predominant factor in determining the issue, as the following facts show :—

1	perforation	while taking food;			operation		$18\frac{1}{2}$	hours after.	Fatal.
1	„	$1\frac{1}{2}$	hours after	„	„		15	„	Recovery.
3	„	2	„	„	1	„	$3\frac{1}{2}$	„	Recovery.
					1	„	20	„	Recovery.
					1	„	56	„	Fatal.
2	„	5	„	„	1	„	2	„	Recovery.
					1	„	$11\frac{1}{2}$	„	Fatal.
1	„	$8\frac{1}{2}$	„	„		„	50	„	Fatal.

An analysis of my whole series shows that *the occurrence of vomiting*, or its absence, has no appreciable influence on prognosis.

Again, it is frequently stated that the *size of the perforation* influences the prognosis. It is assumed that through a large aperture more will escape from the stomach, and the peritoneal infection will be correspondingly rapid and severe. At first sight my observations seem to support this view, because it happens that in four of my gastric cases the perforation was large enough to admit the tip of the little finger, and these all proved fatal ; while in five cases in which the perforation was so small as only to admit the point of a fine probe, all the patients recovered. The influence of the mere size of the opening, however, becomes less significant when it is noticed that in the fatal cases the interval between perforation and operation was 3, 15, $43\frac{1}{2}$, and 96 hours respectively ; while in the cases which recovered it was $4\frac{1}{4}$, 6, $6\frac{1}{2}$, and 23 hours respectively.

In the *duodenal* series also, curiously enough, there were four large perforations, all of which proved fatal ; and four small ones from which recovery took place. Here again, however, the time factor seems to outweigh the mere size of the aperture, for in the large perforations the interval between perforation and operation was $18\frac{1}{2}$, 50, 56, and 60 hours respectively, while in the small ones it was 2, $3\frac{1}{2}$, 15, and 20 hours.

I can suggest no explanation of the unexpected fact that

operation seems to be longer delayed in large perforations than in small ones.

Nor does the *situation of the perforation* appear materially to affect the issue. In all situations—at the cardiac end, in the body of the stomach, or near the pylorus—the cases operated on early gave a much larger proportion of recoveries than those in which the operation was delayed.

The unfavourable influence of *opium* administered for the relief of the early symptoms has already been referred to.

Table IV. Results.

Perforated Gastric *Ulcers.*

(*a*) *Cases operated on within* 12 *hours of perforation*—16.

Recovery.		Fatal.	
No.	Interval between Perforation and Operation.	No.	Interval between Perforation and Operation.
1.	1¾ hours	1.	2 hours
2.	3 „	2.	3 „
3.	4 „	3.	5 „
4.	4¼ „	4.	10 „
5.	6 „		
6.	6 „		
7.	6½ „		
8.	6½ „		
9.	8 „		
10.	10 „		
11.	11 „		
12.	11½ „		

(*b*) *Cases operated on between* 12 *and* 24 *hours of perforation*—6.

Recovery.		Fatal.	
No.	Interval between Perforation and Operation.	No.	Interval between Perforation and Operation.
1.	15 hours	1.	15 hours
2.	21 „	2.	17 „
3.	23 „	3.	18 „

(*c*) *Cases operated on between* 24 *and* 36 *hours of perforation*—6.

Recovery.		Fatal.	
No.	Interval between Perforation and Operation.	No.	Interval between Perforation and Operation.
1.	35 hours	1.	25 hours
2.	36 „	2.	27 „
3.	36 „	3.	36 „

(*d*) *Cases operated on later than* 36 *hours after perforation*—8.

Recovery.		Fatal.	
No.	Interval between Perforation and Operation.	No.	Interval between Perforation and Operation.
		1.	43½ hours
		2.	48 „
		3.	72 „
		4.	96 „
		5.	96 „
		6.	120 „
		7.	120 „
		8.	9 days.

Results.—The accompanying tables (Tables IV., V.) show the results in any series of cases.

In the 36 *gastric cases* (Table IV.) there were 18 recoveries and 18 deaths—an overhead mortality of 50 per cent.

If we deduct those cases in which the patient was evidently dying, and the operation was of the nature of a forlorn hope, say those in which the perforation had existed for over thirty-six hours—and this is considerably beyond the average time allowed by most writers—it leaves 10 fatalities to 18 recoveries, or a mortality of 35.7 per cent.

It will be observed that of the 6 patients operated on between twenty-four and thirty-six hours after perforation, 3 recovered and 3 died ; and of the 6 operated on between twelve and twenty-four hours, 3 recovered and 3 died.

This leaves the cases operated on within twelve hours of perforation—the period within which most writers agree in giving a hopeful prognosis. Of these there were 16, with 12 recoveries and 4 deaths, a mortality of 25 per cent.

There were thus 4 patients who died within the period in which recovery is to be expected, but against these may be placed 6 who survived after being operated upon between fifteen and thirty-six hours from the time of perforation.

The results in the 10 *duodenal cases* are detailed in Table V. Here also the overhead mortality is 50 per cent.

Table V. Results.

Perforated Duodenal *Ulcers.*

(*a*) *Cases operated on within* 12 *hours of perforation*—3.

Recovery.		Fatal.	
No.	Interval between Perforation and Operation.	No.	Interval between Perforation and Operation.
1.	2 hours	1.	11½ hours
2.	3½ „		

(*b*) *Cases operated on between* 12 *and* 24 *hours of perforation*—3.

Recovery.		Fatal.	
No.	Interval between Perforation and Operation.	No.	Interval between Perforation and Operation.
1.	15 hours	1.	18½ hours
2.	20 „		

(*c*) *Cases operated on over* 24 *hours after perforation*—4.

Recovery.		Fatal.	
No.	Interval between Perforation and Operation.	No.	Interval between Perforation and Operation.
1.	14 days (Operation for sub-phrenic abscess.)	1.	50 hours
		2.	56 „
		3.	60 „

So far as I have been able to find, the above is the largest individual experience hitherto reported, and it is interesting to notice that the percentage mortality corresponds as closely as

could be expected, considering the disparity in numbers, with the results in 237 cases of perforated gastric ulcer collected by F. and G. Gross. These are their figures: operated on within 12 hours, 25 per cent. mortality; between 12 and 24 hours, 52.7 per cent.; between 36 and 48 hours, 56 per cent.; over 48 hours, 73 per cent.; giving a general mortality of 47.26 per cent.

I would not seek to attach great importance to tables of mortality in considering such operations of urgency as those with which we are now concerned. In the nature of things the death-rate from perforation of the alimentary canal must always remain comparatively high. That it will yet be reduced far below the point at which it now stands I have no doubt, but I would plead that this reduction be effected by operating earlier and oftener in such cases, and by improving our methods, and not by what is euphemistically spoken of as "carefully selecting our cases." In submitting the above record, I would ask the Society to bear in mind that I have never refused to operate on a patient on account of the gravity of his condition. Several of the patients were obviously moribund, and were only operated upon to afford what seemed the last remaining chance. Nothing was promised, little expected, hope alone remained, and if I am encouraged to continue to operate on such patients in the last extremity, it is because in more than one case this course has been justified by results.

Summary of Results.—In Table VI. I have summarised the results of the combined gastric and duodenal cases, showing the percentage mortality at intervals of twelve hours between perforation and operation.

TABLE VI. SUMMARY OF RESULTS.

Gastric cases,	36	18 Recoveries	18 Fatal.
Duodenal cases,	10	5 „	5 „
Total,	46	23	23

	Total.	Recovery.	Fatal.	Mortality.
Operated on within 12 hours of perforation	19	14	5	26.3%
„ between 12 and 24 hours of perforation	9	5	4	44.4%
„ „ 24 and 36 „	6	3	3	50%
„ later than 36 „	12	1	11	91.7%
Total . . .	46	23	23	50%

No useful purpose is served, I find, by drawing comparisons between these results and those of other individual observers. The limited number of cases which falls to any single surgeon renders the figures available for purposes of statistical comparison useless.

Perforated Gastric and Duodenal Ulcers.

Summary.—The main conclusions to be drawn from a study of these cases may be summarised as follows :—

1. The liability to perforation appears to be as great in the male as in the female, as, in spite of the greater frequency with which women suffer from ulcer, 20 of my patients were males, and 26 females.
2. On the whole, perforation occurs at an earlier age in females than in males; and, other things being equal, the younger the patient is, the better seems to be the prognosis.
3. While perforation occasionally occurs in patients who have never hitherto suffered from any digestive disturbance, there is in the great majority of cases a previous history of severe indigestion, and in a considerable number, of ulceration of the stomach or duodenum. Hæmatemesis, however, is a rare antecedent symptom.
4. In a considerable number of my cases symptoms premonitory of perforation were present.
5. No constant factor which would account for the actual rupture of the ulcer can be traced.
6. The site of the *gastric* perforations was on the anterior wall of the stomach in 33 cases, and on the posterior wall in 3. Of these 4 were at the cardiac end; 14 in the body of the stomach; and 18 towards the pyloric end. Practically all were towards the lesser curvature. All the *duodenal* perforations were in the first part of the duodenum, and, with one exception, on the anterior wall.
7. Only in a single case was there more than one perforation.
8. The size of the perforations varied from that of a pin's head to that of a three-penny piece. In two cases it was in the form of a longitudinal tear. I cannot confirm the statement commonly made, that the acuteness of onset, the severity of the symptoms, and the degree and extent of the ensuing peritonitis bear any constant relationship to the size of the perforation.
9. The most characteristic clinical features of perforation are the suddenness of onset and the overpowering nature of the pain; the marked abdominal rigidity; and the severe degree of shock.

10. Vomiting is not a common symptom at the onset; it occurred only in 12 out of 37 cases in which the point was noted. I find no evidence for the statement that the occurrence of vomiting increases the quantity of fluid which escapes into the peritoneal cavity, or adds to the gravity of the prognosis.
11. The information derived by percussion is inconstant and unreliable. Early absence or impairment of the liver dulness is suggestive of perforation; its disappearance later is of no diagnostic value. Persistence of the normal hepatic dulness does not negative a diagnosis of perforation.
12. The situation of the maximum area of pain and of tenderness to pressure forms a reliable guide to the seat of the perforation, and therefore to the site of the incision.
13. The apparent improvement which accompanies the reaction from the initial shock is very deceptive, and frequently accounts for fatal delay in operating.
14. The risk involved in administering opium for the relief of the initial symptoms is illustrated by these figures: in 22 cases no opium was given, 5 patients died, 17 recovered; in 12 cases opium was given, 10 patients died, 2 recovered.
15. There is no constant relationship traceable between the severity of the peritonitis which ensues, and the size or site of the perforation, the quantity or quality of the fluid which escapes from the stomach, the state of repletion of the stomach at the moment of perforation, or any other single factor. Food particles are very seldom recognisable in the fluid found in the peritoneal cavity.
16. In all cases the abdomen should be opened as early as possible, by a vertical incision, passing between the fibres of the rectus, and usually placed over the site of maximum tenderness.
17. It is seldom, if ever, necessary or advisable to open the stomach to find the perforation.
18. An omental graft should be fixed over the line of sutures occluding the perforation, to add to the security of the closure. Leakage is extremely rare.
19. Excision of the ulcer is seldom necessary to secure occlusion of the perforation, and, unless absolutely essential for this purpose, is not to be recommended.
20. In comparatively few cases is the patient in a condition to justify gastro-enterostomy being performed merely as a precautionary measure against stenosis of the pylorus, or as an additional means of favouring healing of the ulcer. If the closure of the ulcer has actually resulted in serious narrowing of the pylorus, gastro-enterostomy should be performed.
21. In certain cases pyloroplasty is a simple and efficient alternative to gastro-enterostomy.

22. The abdomen should usually be washed out, a special opening being made above the pubis for the purification of the pelvis and for purposes of drainage. To favour drainage the head of the bed should be raised.
23. The free use of saline injections is most beneficial; fluids may be given by the mouth after twenty-hours.
24. The after-complications I have met with have been temporary gastric fistula, pneumonia, bronchitis, acute non-suppurative parotitis, phlebitis of the leg, pelvic abscess, sub-phrenic abscess.
25. The predominant factor in determining the prognosis is the period which is allowed to elapse between the occurrence of perforation and the performance of operation. Every hour of delay diminishes the patient's chance of recovery.
26. The results in my 46 cases are summarised in Table VI. (page 322).

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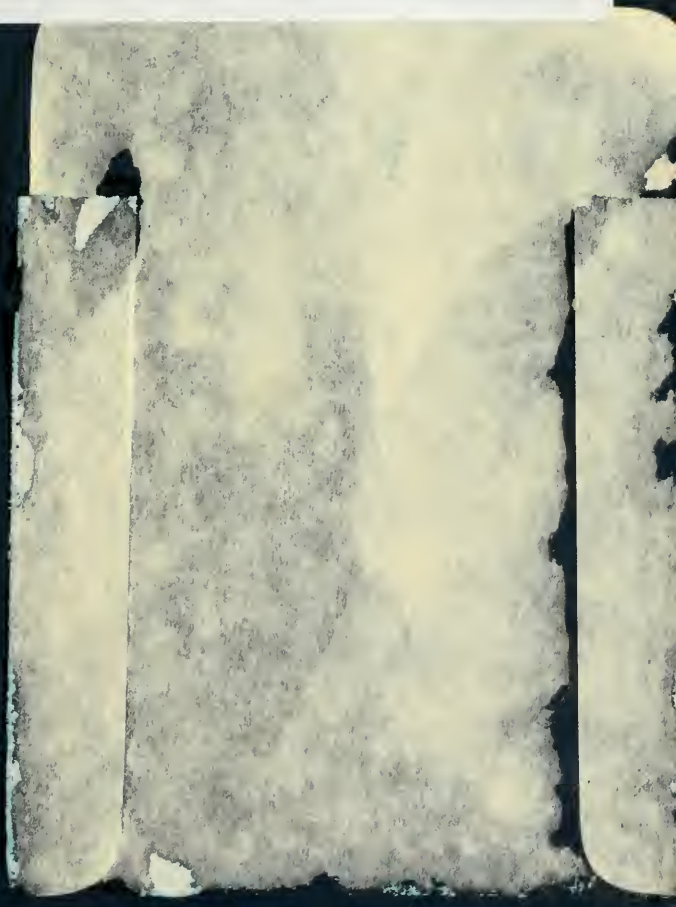
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